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FACTOR ANALYSIS OF THE DISABILITIES OF THE ELDERLY WITH THE AID OF AN ELECTRONIC CALCULATOR

BY

JOHN MADDISON, R. C. F. SMITH, AND O. OLBRICH

Twickenham, Middlesex *Boldon, Co. Durham* *Sunderland*

There have been a number of surveys of old people by doctors (Walker, 1947; Sheldon, 1948; Adams and Cheeseman, 1951; Simonds and Stewart, 1954), but no survey that we know of has attempted full medical examination of a sample in a localized community.* Early in 1952 we thought it would be a useful piece of work to determine accurately the diseases and disabilities of a sample of the men and women 60 years of age and over in a circumscribed area. We chose the villages of Tunstall and Silksworth in Co. Durham, which together are typical of a mixed urban community with an average normal healthy population and with housing conditions and wages just a shade higher than average. The estimated population is 7,500; the acreage is 1,720; there are 2,300 houses; the number of persons per house is a fraction over 3 but under the expected average of 3½. These conditions are a little better than usual, but to offset this there are several small groups of miners' houses with twelve houses to the acre, which are small and poorly built. For the whole area the death rate and incidence of infectious disease are about the same as for England and Wales. The workers are employed mostly in the coal mining and shipping industries and in small shops.

Arranging the Medical Examinations.—The names and addresses of all people aged 60 and over were obtained by using every source of information possible, such as the voting lists, organizations and clubs, churches, welfare officers, and medical practitioners. The completed list contained 550 names, or 7·3 per cent. of the population, which is slightly less than that expected in comparable areas of England and Wales. All the local practitioners offered to encourage the old people they met in their practices to attend for examination.

By courtesy of Dr. O. Olbrich, consulting physician in charge of geriatrics at Sunderland General Hospital, the facilities for the medical examinations in his department were placed at Dr. Smith's disposal. He arranged for about sixty people from the list to be invited each week to attend the hospital. About half of them accepted; but some feeling quite well and not in need of medical attention, some fearing something being found out, some saying they did not wish to tempt providence, some recently attended by their own doctors or already attending hospital, some being ill in bed, and some for other reasons, were reluctant or refused to attend. These Dr. Smith visited and in this way more were persuaded to come. Some private-car owners, including the Red Cross, volunteered to provide transport. The number which finally attended was 238 out of the original 550; the sample is obviously biassed.

The medical examinations were very thorough, covering every system of the body and including *x* rays and laboratory tests. Dr. Smith did much of the clinical work himself; he was assisted by four other doctors in the geriatric department, a sister, two nurses, and a clerk. The findings for each person examined were entered on a separate form specially designed to ensure that nothing was overlooked.

Analysis of the Data.—The technique of factor analysis can be used as a tool for probing medical data to see if any factors or common influences can be detected to account for the individual differences in the people examined, and as a method of classification (Maddison, 1953).

We decided to try a factor analysis of the clinical findings of our sample. This begins with a Table showing the correlation between every pair of attributes. In our investigation some of the observations such as blood pressure took the form of measurements, but most of them were qualitative; that is the observation showed whether or not a person suffered from a disease, there being no question of degree. The first step in the analysis was the transformation of the qualitative observations into correlations. Burt (1950) has shown how this is done. For the benefit of readers who are not well acquainted with the methods of factor analysis, we have given a simplified account of the procedures used in this part of the paper. Full accounts are given by Thurstone (1947), Thomson (1950), and Cattell (1952).

FACTOR ANALYSIS

TABULATION.—We selected certain "determinables" from the clinical records of the 238 old persons examined; these were sex, age, percentage of

* Since this was written there have appeared Hobson and Pemberton (1955), and Exton-Smith (1955).

haemoglobin, systolic blood pressure, diastolic blood pressure, and diseases found. Some persons had two diseases, some three, and a few four; we therefore arranged the diseases for each person in order of importance: first, second, third, and fourth. The complete list of possible diseases is set out in Table I, which also gives the number of times each one was noted.

TABLE I

DISEASES AND NUMBER OF TIMES FOUND

	Diseases Found	Number of Times each Disease was Found (order of importance)									
		1st		2nd		3rd		4th		Total	
		Men	Women	Men	Women	Men	Women	Men	Women	Men	Women
Infective Diseases	Poliomyelitis; old paralysis	—	—	—	1	—	—	—	—	—	1
	Shingles	—	—	1	—	—	—	—	—	1	—
Neoplasms	Carcinoma of colon	1	—	—	—	—	—	—	—	1	—
	Carcinoma of breast	—	1	—	—	—	—	—	—	—	1
	Carcinoma of prostate	2	—	—	—	—	—	—	—	2	—
	Lipoma	—	—	—	1	—	—	—	—	—	1
Allergic, Endocrine, Metabolic and Nutritional Diseases	Vasomotor rhinorrhoea	—	1	—	—	—	—	—	—	—	1
	Asthma	—	3	—	1	—	—	—	—	—	4
	Myxoedema	—	2	—	1	—	—	—	—	—	3
	Diabetes	1	2	1	2	—	1	—	—	2	5
	Adenoma of pituitary	—	1	—	—	—	—	—	—	—	1
	Adiposity; obesity	1	6	2	16	2	4	—	3	5	29
Blood Diseases	Anaemia pernicious	—	4	1	1	—	—	—	—	1	5
	Anaemia microcytic	—	5	—	1	—	1	—	1	—	8
Psychoneurotic Disease	Anxiety state	—	3	—	1	—	—	—	—	—	4
Diseases of Nervous System and Sense Organs	Cerebral embolism and thrombosis	1	—	1	1	—	—	—	—	2	1
	Multiple sclerosis	1	—	—	—	—	—	—	—	1	—
	Arterio-sclerotic Parkinsonism; paralysis agitans	2	—	1	—	—	—	—	—	3	—
	Cerebral paralysis; paraplegia; hemiplegia, upper motor neuron lesion	1	—	—	2	—	—	—	—	1	2
	Epilepsy	—	1	—	1	—	—	—	—	—	2
	Neuralgia—trigemminal nerve	—	—	—	—	—	1	—	—	—	1
	Paralysis	—	1	—	—	—	—	—	—	—	1
	Choroiditis	—	—	1	—	—	—	—	—	1	—
	Cataract	—	1	2	1	—	1	—	—	2	3
	Coloboma	1	—	—	—	—	—	—	—	1	—
	Blindness both eyes	1	1	—	—	—	—	—	—	1	1
	Blindness one eye	—	—	1	—	—	—	—	—	1	—
	Otitis media; otorrhoea—chronic	—	1	2	—	—	—	—	—	2	1
	Vertigo	1	1	—	—	—	—	—	—	1	1
	Oto-sclerosis	1	—	—	—	—	—	—	—	1	—
	Deafness	2	—	—	—	—	2	—	1	2	3
Diseases of Circulatory System	Angina	3	5	2	1	—	—	—	—	5	6
	Mitral stenosis; early heart failure	—	3	1	—	—	—	—	—	1	3
	Endocarditis—chronic—aortic valve	1	—	—	—	—	—	—	—	1	—
	Auricular fibrillation	1	1	—	—	—	—	—	—	1	1
	Congestive heart failure	1	—	—	1	—	—	—	—	1	1
	Left ventricular failure	1	2	—	1	—	—	—	—	1	3
	Enlarged heart; enlarged left ventricle; corpulmonale	1	1	5	2	—	—	—	—	6	3
	Hypertension benign	8	19	1	1	—	—	—	—	9	20
	Hypertension malignant	9	21	1	1	—	—	—	—	10	22
	Hypertension systolic	3	6	—	—	—	—	1	—	4	6
	Arteriosclerosis; diffuse hyperplastic sclerosis; atheroma; calcified aorta	1	1	1	1	1	—	—	—	3	2
	Aneurysm—fusiform	1	—	1	—	—	—	—	—	2	—
	Intermittent claudication	—	—	—	1	—	1	—	—	—	2
	Atheroma—unfolded aorta	—	—	1	—	1	—	—	—	2	—
	Dermatitis—varicose; varicose veins; varicose ulcer	—	3	2	2	—	1	—	—	2	6
	Haemorrhoids; piles	—	—	—	—	1	2	—	—	1	2
Diseases of Respiratory System	Tracheitis—acute	—	1	—	—	—	—	—	—	—	1
	Bronchitis acute	1	1	—	—	—	—	—	—	1	1
	Bronchitis chronic	4	11	4	3	1	—	—	—	9	14
	Sinusitis	—	—	—	—	—	—	—	1	—	1
	Pneumononoconiosis	5	—	—	—	—	—	—	—	5	—
	Interstitial pneumonia; fibrosis of lung	1	—	—	—	—	—	—	—	1	—
	Bronchiectasis	—	1	—	—	—	—	—	—	—	1
	Emphysema	6	2	2	1	—	—	—	—	8	3

contin.—

TABLE I—*continued*

Diseases Found		Number of Times each Disease was Found (order of importance)									
		First		2nd		3rd		4th		Total	
		Men	Women	Men	Women	Men	Women	Men	Women	Men	Women
Diseases of Digestive System	Gingivitis; septic gums	—	—	—	—	—	1	—	—	—	1
	Pyloric stenosis	—	1	—	—	—	—	—	—	—	1
	Hernia—inguinal	3	1	—	—	—	—	—	—	3	1
	Hernia—ventral	—	—	—	1	—	—	—	—	—	1
	Diverticulitis	—	1	—	—	—	—	—	—	—	1
	Constipation	—	—	—	1	—	—	—	—	—	1
	Colon—spastic	—	1	—	—	—	—	—	—	—	1
	Fistula—rectum	1	—	1	—	—	—	—	—	2	—
	Cholecystitis	—	3	—	2	—	3	—	—	—	8
Diseases of Genito-Urinary System	Pyelitis	—	—	1	—	—	—	—	—	1	—
	Calculi of kidney and ureter; renal calculus	1	1	—	—	—	—	—	—	1	1
	Cystitis	1	—	—	—	—	1	—	—	1	1
	Caruncle of urethra	—	1	—	—	—	—	—	—	—	1
	Prostate—enlargement	3	—	5	—	3	—	1	—	12	—
	Phimosis	1	—	—	—	—	—	—	—	1	—
	Cystocyle; stress incontinence; uterine prolapse	—	3	—	5	—	2	—	—	—	10
Diseases of Skin	Dermatitis (unqualified)	—	—	—	1	—	—	—	—	—	1
Diseases of Bones and Joints	Rheumatoid arthritis	—	—	—	1	—	1	—	—	—	2
	Osteo-arthritis	3	2	—	6	1	4	1	1	5	13
	Fibrositis	—	2	—	3	—	—	—	—	—	5
	Displacement of intervertebral disk	—	—	—	1	—	—	—	—	—	1
	Bunions	—	—	—	1	—	—	—	—	—	1
	Synovitis—non-occupational	—	—	—	1	—	—	—	—	—	1
	Synovitis—occupational; bursitis	—	1	—	—	—	—	—	—	—	1
	Kyphosis; lumbar lordosis	—	—	—	1	—	1	—	—	—	2
	Flat feet	1	—	—	—	—	—	—	—	1	—
Congenital Malformations	Congenital heart disease; dextra cardia; situs inversus of liver	—	1	—	—	—	—	—	—	—	1
	Ichthyosis	—	—	—	—	—	—	1	—	1	—
	Congenital deformity—foot	—	—	—	—	1	—	—	—	1	—
Symptoms and Ill-defined Conditions	Insomnia	1	—	1	—	—	—	—	—	2	—
	Sympathetic block	—	—	—	1	—	—	—	—	—	1
	Backache—chronic	1	—	—	—	—	—	—	—	1	—
	Headache	—	1	—	2	—	—	—	1	—	4
Total		79	131	42	72	11	27	4	8	136	238

Each determinable was divided into a number of subclasses or attributes. To reduce the number of attributes to manageable size, some items with small frequencies were pooled; the 42 attributes finally chosen are set out in Table II (overleaf).

Each attribute was given a code number, those for diseases being taken from the W.H.O. International Classification (1948). The information about each person was then recorded in code form on a punch card for use with Hollerith machinery. The set of cards for the 238 persons, each with appropriate details of the 42 attributes, constituted a Table showing the presence or absence of the attributes for the given sample of persons. If the presence of an attribute is denoted by one, and its absence by nought, the Table will appear as in the sample illustration Table III (overleaf).

FREQUENCY TABLE.—The cards were sorted on a machine into groups and subgroups so as to form a Table of frequency of persons, characterized by the attributes specified, taken in pairs. The Table contained 42 × 42 entries: the top left-hand corner is shown in Table IV (overleaf).

Table IV can be obtained by multiplying each element in each row of Table III by the corresponding element in each column in turn of the same Table when transposed, that is with the rows arranged as columns (Table IIIA). The products thus formed are summed and entered in turn in the cells of each row of Table IV. The product-sum formed by Row j of Table III and Column k of Table IIIA is always entered in the cell at the junction of Row j and Column k of Table IV.

TABLE II

DETERMINABLES, ATTRIBUTES, AND FREQUENCIES IN PERSONS

Determinable		Attribute	Attribute Reference Number	Number of Persons	
				Men	Women
Sex		Man	1	90	—
		Woman	2	—	148
Age Group		60–64	3	19	59
		65–69	4	28	41
		70–74	5	25	28
		75–79	6	14	13
		80–84	7	4	7
Percentage of Haemoglobin		30–69	8	7	17
		70–79	9	13	47
		80–89	10	30	41
		90–99	11	20	26
		100–129	12	20	17
Blood Pressure	Systolic	130–159	13	22	15
		160–179	14	26	36
		180–199	15	16	34
		200–219	16	15	24
		220–239	17	8	23
		240–299	18	3	16
	Diastolic	60–89	19	27	22
		90–99	20	27	40
		100–109	21	13	38
		110–119	22	10	25
		120–159	23	13	23
Most Important Disease	First	Nil	24	12	16
		Ineffective, neoplasm, allergic	25	5	17
		Anaemia	26	—	9
		Mental, nervous, senses	27	11	9
		Heart, arteries	28	10	16
		Benign hypertension	29	11	25
		Malignant hypertension	30	9	21
		Respiratory	31	17	16
		Digestive, genito-urinary	32	9	12
		Skin, bone, malformation, etc.	33	6	7
	Second	Nil	34	46	78
		Infective, neoplasm, allergic	35	4	20
		Anaemia, mental, nervous, senses	36	9	8
		Heart, arteries, hypertension	37	15	11
		Respiratory, digestive, genito-urinary, etc.	38	16	31
	Third	Nil	39	79	121
		All diseases	40	11	27
	Fourth	Nil	41	86	140
		All diseases	42	4	8

TABLE III

ATTRIBUTES OF PERSONS

Determinable		Attribute	Persons (on punch cards)								
			A	B	C	D	E	F	G		
Sex		Man	1	0	1	0	0	0	1	.	.
		Woman	0	1	0	1	1	1	0	.	.
Age Group		60–64	1	0	1	0	1	0	0	.	.
		65–69	0	1	0	1	0	0	1	.	.
		.	.	.	.	.	.	.	.	.	.
Most Important Disease	First	None Found	0	0	1	0	1	0	0	.	.
		Respiratory	1	0	0	0	0	0	1	.	.
		Hypertensive	0	1	0	0	1	0	0	.	.
		Allergic	0	0	0	0	0	1	0	.	.
		.	.	.	.	.	.	.	.	.	.
	Second	None Found	1	0	1	0	1	0	0	.	.
		Digestive	0	1	0	1	0	0	1	.	.
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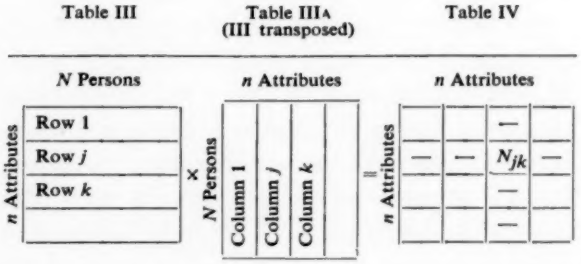
Thus in diagrammatic form:



and in symbols: post-multiply Table III by its transpose Table IIIA to give Table IV.

$$\text{III} \times \text{III}_\text{A} = \text{IV} \quad \ldots \quad \ldots \quad \ldots \quad \ldots \quad (i)$$

TABLE IV
FREQUENCY TABLE OF ATTRIBUTES

		Sex		Age Group					Haemoglobin					Systolic Blood Pressure					
		Men	Women	60–64	65–69	70–74	75–79	80–84	30–69	70–79	80–89	90–99	100–129	130–159	160–179	...	...	...	...
Sex	Men	90	—	19	28	25	14	4	7	13	30	20	20	22	26	..	..	..	..
	Women	—	148	59	41	28	13	7	17	47	41	26	17	15	36	..	..	..	..
Age	60–64	19	59	78	—	—	—	—	7	23	23	11	14	17	24	..	..	..	..
	65–69	28	41	—	69	—	—	—	5	18	19	17	10	9	19	..	..	..	..
	70–74	25	28	—	—	53	—	—	6	9	19	11	8	8	13	..	..	..	..
	75–79	14	13	—	—	—	27	—	5	6	8	5	3	2	6	..	..	..	..
	80–84	4	7	—	—	—	—	11	1	4	2	2	2	1	—	..	..	..	..
Haemo-globin	30–69	7	17	7	5	6	5	1	24	—	—	—	—	1	9	..	..	..	..
	70–79	13	47	23	18	9	6	4	—	60	—	—	—	11	14	..	..	..	..
	80–89	30	41	23	19	19	8	2	—	—	71	—	—	14	22	..	..	..	..
	90–99	20	26	11	17	11	5	2	—	—	—	46	—	4	11	..	..	..	..
	100–129	20	17	14	10	8	3	2	—	—	—	—	37	7	6	..	..	..	..
Systolic Blood Pressure	130–159	22	15	17	9	8	2	1	1	11	14	4	7	37	—	..	..	..	..
	160–179	26	36	24	19	13	6	—	9	14	22	11	6	—	62	..	..	..	..
	... ...	..	..	..	..	..	..	..	..	..	..	..	.	..	..	..	..	..	..
	... ...	..	..	..	..	..	..	.	.	..	..	..	.	..	..	..	..	..	..
	... ...	..	..	..	..	..	..	.	.	..	..	..	.	..	..	..	..	..	..

TABLE V
ATTRIBUTES COMMON TO EACH PERSON

Determinates	29 Persons											.	.	.	29	Total
	1	2	3	4	5	6	7	8	9	10	11					
Row *j* from III Men	1	0	1	0	0	0	1	0	1	0	0	.	0	.	0	4 = N_j; *i.e.* three men with respiratory disease and one without
Column *k* from IIIA Respiratory Disease	1	1	0	1	1	1	1	1	1	1	0	.	0	.	0	9 = N_k; *i.e.* three respiratory diseases in men and six not men
Cross Products	1	0	0	0	0	0	1	0	1	0	0	.	0	.	0	3 = N_{jk}; *i.e.* three men with respiratory disease; entry in a cell of Table IV or VI.

Suppose one Row *j* of persons from Table III and a different Column *k* of persons from Table IIIA were arranged as in Table V, we should have the result shown in Table V (above).

The third row of Table V (Cross Products) gives the attributes possessed in common by each person; these entries are also the cross products between row and column. The totals such as N_{jk} form the entries in the cells of tables like Table IV or Table VI (overleaf).

FORMING THE CORRELATIONS.—A correlation is a ratio. It provides a measure of the amount of association between two sets of observations on a series of persons or things. If the correlation is one there is perfect association; if it is nought there is no association; and if it is something in between, *e.g.* 0·5, there is partial association. The correlation is obtained by finding the geometric average (that is square root of product) of two slopes called regression lines.

Suppose the observations are plotted on a graph with axes *X* and *Y* (Fig. 1, overleaf). One regression line (*y* on *x*) tells us the average amount of increase up the *Y* axis for each unit length along the *X* axis; the other regression line (*x* on *y*) tells us the average amount of increase along the *X* axis for each unit up the *Y* axis. The geometric mean of these two is the correlation.*

Each regression slope is not a simple average of the slopes of single points. It is itself a ratio: the numerator is an area—the sum of the rectangles formed by the product from each *x* and *y* value. The denominator is an area—the sum of the squares formed from the *x* values, or the sum of the squares formed from the *y* values.

The correlation may be expressed thus in words:

$$\frac{\text{Sum of Cross Products}}{\sqrt{(\text{Sum of Squares on } X \text{ Axis})\ (\text{Sum of Squares on } Y \text{ Axis})}}$$

and in symbols thus:

$$\frac{N_{jk}}{\sqrt{N_j \cdot N_k}} = \sqrt{\frac{N_{jk} \cdot N_{jk}}{N_j \cdot N_k}} \quad \ldots \quad \ldots \quad (ii)$$

where N_{jk} is an entry in a side cell of Table IV, and N_j, N_k are the entries in the respective row and column of the diagonal.

* We are deliberately avoiding the complication in the usual computation of correlations in which the sums of cross products and squares are measured from the mean.

Suppose the totals of Row j and Column k from Table V were plotted on a graph as in Fig. 1, then:

Product of one occurrence "man" (x) × one occurrence "respiratory disease" (y)	$= 1 \times 1 = 1$		
Sum of three products (xy) for three occurrences "men with respiratory disease"	$= 3 \times 1$	$= 3$ ...	N_{jk}
Square for one man (x)	$= 1 \times 1 = 1$		
Sum of four man squares (x) for four men	$= 4 \times 1$	$= 4$ ...	N_j
Square of one respiratory disease (y)	$= 1 \times 1 = 1$		
Sum of nine respiratory disease squares (y)	$= 9 \times 1$	$= 9$ ...	N_k
Slope or regression (of y on x) rise of y for 1 x		$= 3/4$ ...	N_{jk}/N_j
Slope or regression (of x on y) increase of x for 1 y		$= 3/9$ ...	N_{jk}/N_k
Geometric mean = Square root of $3/4 \times 3/9 = \sqrt{9/36} \cdot \sqrt{1/4}$		$= 1/2$ ...	$\sqrt{N_{jk} \cdot N_{jk}/N_j \cdot N_k}$

Suppose for example we make a small table (Table VI), which is like Table IV but complete. The correlations formed by applying Equation (*ii*) would then be as in Table VII.

In Table VII, first the rows of Table VI and then the resulting columns are divided by the square root of the number in the appropriate corresponding diagonal cell. Thus the entry 3 opposite Respiratory and under Men is divided by $\sqrt{9}$, *i.e.* 3, and then by $\sqrt{4}$, *i.e.* 2. The result is 0·500 in Table VII. This procedure converts each diagonal cell of Table VII to unity and makes the entries in the side cells of relative comparability. The rows and columns of Table IV were treated in this way; the answers were found by machinery and punched on cards in the binary scale ready for the next operation.

TABLE VI
FREQUENCIES

		Sex		Disease		
		Men	Women	None Found	Respiratory	Hypertensive
Sex	Men	4	0	0	3	1
	Women	0	25	4	6	15
Disease	None Found	0	4	4	0	0
	Respiratory	3	6	0	9	0
	Hypertensive	1	15	0	0	16

TABLE VII
CORRELATIONS

		Sex		Disease		
		Men	Women	None Found	Respiratory	Hypertensive
Sex	Men	1·000	·000	·000	·500	·125
	Women	·000	1·000	·400	·400	·750
Disease	None Found	·000	·400	1·000	·000	·000
	Respiratory	·500	·400	·000	1·000	·000
	Hypertensive	·125	·750	·000	·000	1·000

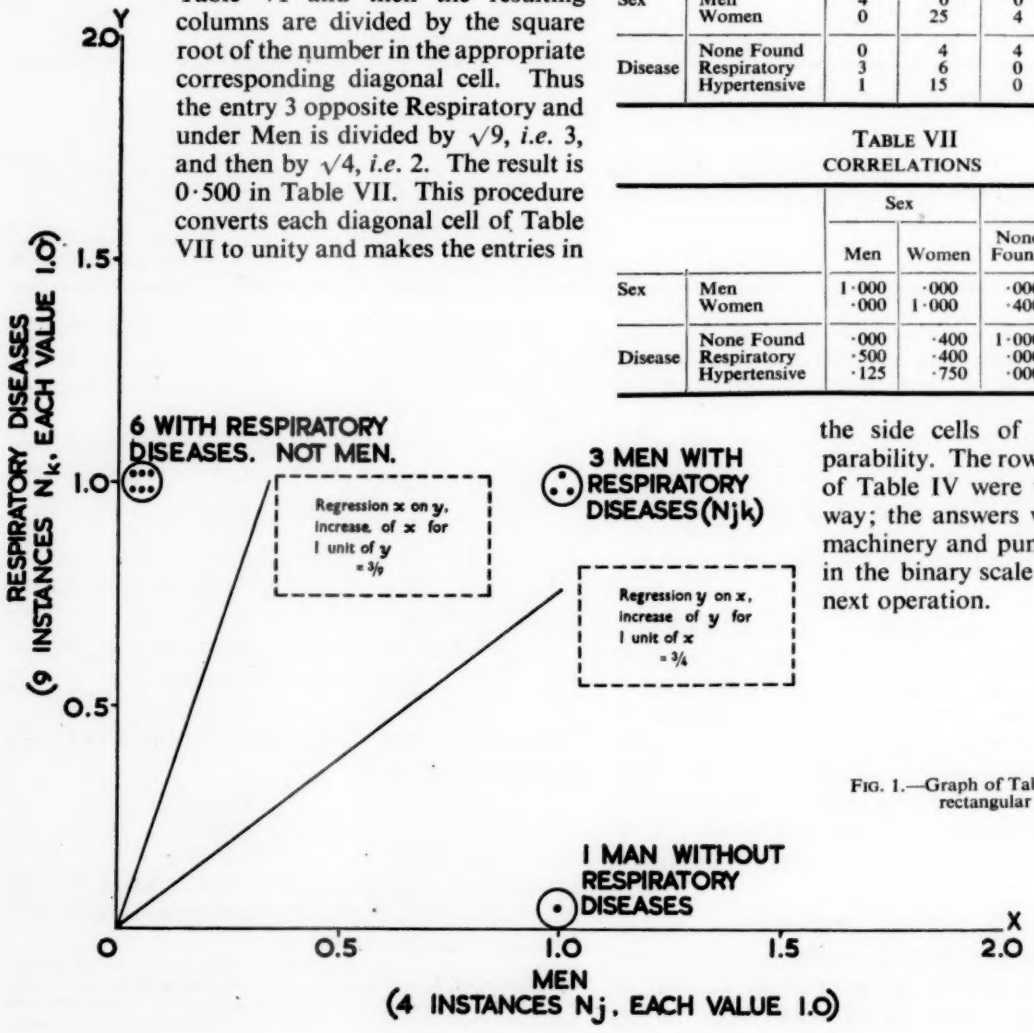


FIG. 1.—Graph of Table V plotted on rectangular axes.

TABLE VIII

NORMALIZED VALUES

Determinates	29 Persons															Total	Sum of Squares for Each Row
	1	2	3	4	5	6	7	8	9	10	11	.	.	.	29		
Row *j* from III Men	$\frac{1}{3}$	0	$\frac{1}{3}$	0	0	0	$\frac{1}{3}$	0	$\frac{1}{3}$	0	0	.	0	.	0	2	1
Column *k* from IIIA Respiratory Disease	$\frac{1}{3}$	$\frac{1}{3}$	0	$\frac{1}{3}$	$\frac{1}{3}$	$\frac{1}{3}$	$\frac{1}{3}$	$\frac{1}{3}$	$\frac{1}{3}$	$\frac{1}{3}$	0	.	0	.	0	3	1
Cross Products	$\frac{1}{9}$	0	0	0	0	0	$\frac{1}{9}$	0	$\frac{1}{9}$	0	0	.	0	.	0	3/6 = $\frac{1}{2}$	

THE COSINE CONVENTION.—In Table III we gave a value one to each person who possessed an attribute and the value nought when he did not. The following step is simplified if we regard the correlations as having been obtained in a different way. In Table V, Row *j* (men) contains four values each of one making a total of four. Each value one^2 is one, so that the sum of the squares is 4 and its square root 2. If we divide each value one by this square root we get four values each $\frac{1}{2}$. The values are now said to be "normalized". Similarly the second Row *k* (respiratory disease) has nine values of one unit = total 9. The root of the sum of the square values is 3. Each "normalized" value becomes $\frac{1}{3}$. The normalized values for Table V now become as in Table VIII.

The sum of the squared values for each row of Table VIII is one. Thus, when normalized values are used in Equation (*ii*), the denominator becomes unity; the numerator—the sum of the normalized cross products—becomes equal to the correlation; in our example the correlation is $\frac{1}{2}$.

If now we prepare a graph (Fig. 2) in which the axes, instead of being at right angles, are placed at an angle whose cosine is equal to the correlation coefficient, then, when we plot the normalized entries from Table VIII, the sum of the areas formed by the cross products is zero.

In Fig. 2 the axes *OX* and *OY* are placed at an angle of 60° whose cosine is $\frac{1}{2}$ to correspond with the correlation from Table VIII. The area *OBCD* is that formed by the normalized cross products. The formula for this area, which consists of two right-angled triangles *OBC* and *ODC* put together is:

$$\frac{OB \cdot OD}{\sin DOB} - \frac{(OB^2 + OD^2) \cot DOB}{2} \quad \ldots (iii)$$

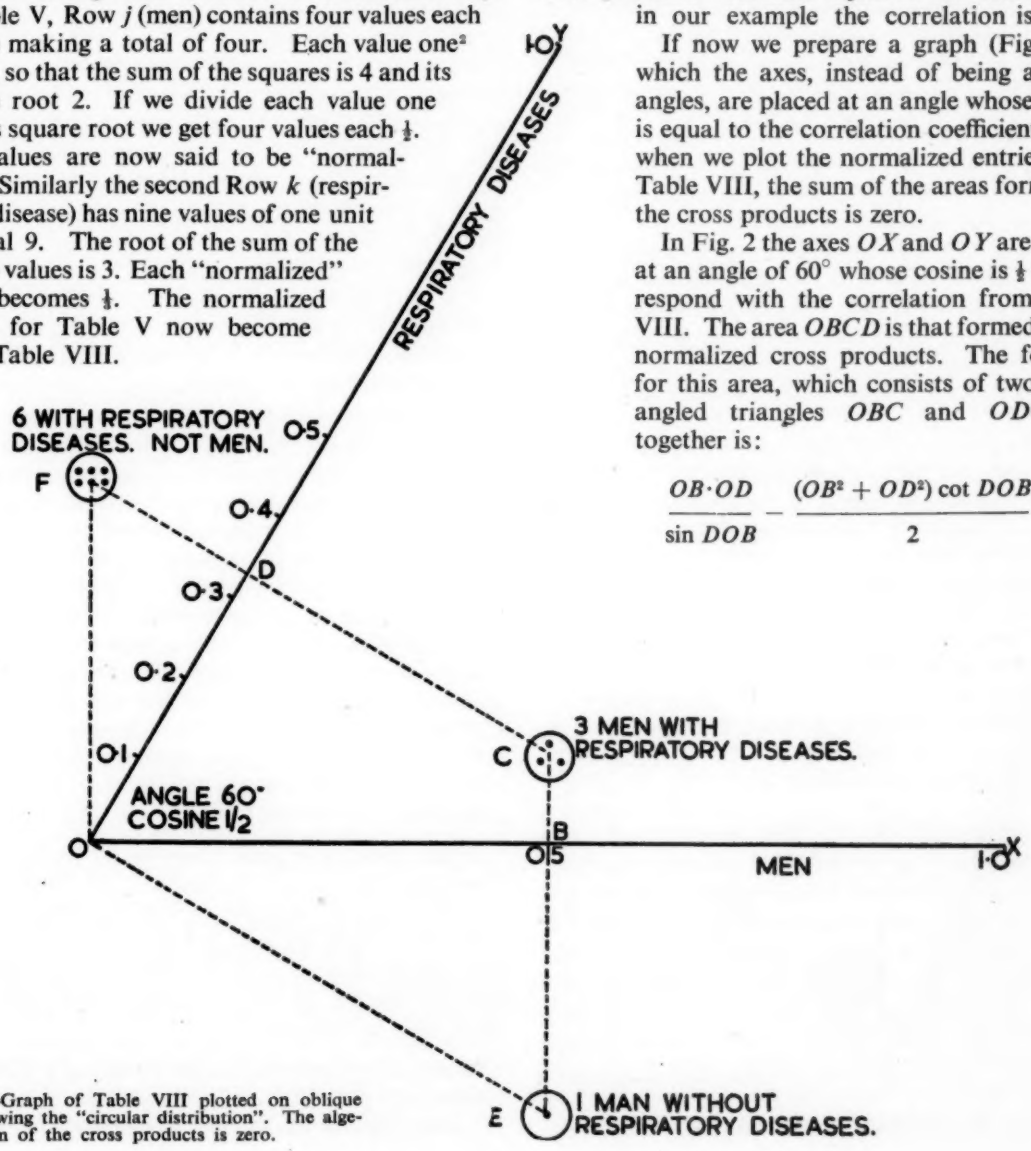


FIG. 2.—Graph of Table VIII plotted on oblique axes showing the "circular distribution". The algebraic sum of the cross products is zero.

Substituting the numerical values, we obtain 0·2646. Similarly, area *OEB* which represents one "man-not-respiratory" is − 0·0722; and area *ODF* which represents six "respiratory-not-men" is − 0·1924. The two minus areas exactly balance the plus area so that the algebraic sum is zero.

The diagram (Fig. 2) illustrates the use of two axes *X* and *Y* placed so that the angle between them has a cosine equal to the correlation between the two attributes—men and respiratory disease. This correlation appears in the appropriate side cell at the junction of a row and a column of Table VII. Every side cell can be similarly represented by a pair of axes at an angle. In three dimensions they are like the edges of a three-cornered pyramid standing on its point. In many dimensions they have to be imagined; but we may in the meantime think of some of them arranged like the ribs of a half-opened umbrella or pins in a pin cushion.

Thus, by using the convention of plotting persons on a graph with axes at an angle whose cosine is equal to the correlation coefficient, we get the association of the persons on two attributes into a circular distribution and in geometrical balance. The two axes with their appropriate angle form a pair of vectors which summarize the distribution of the persons, and which can be used to represent the persons in diagrammatic form on paper and in space.

RESOLVING THE VECTORS.—If we draw a line, *OX* say (Fig. 3), on a sheet of paper, we can give it a certain direction and a certain length; it then becomes a vector. From one end of the line, say O,

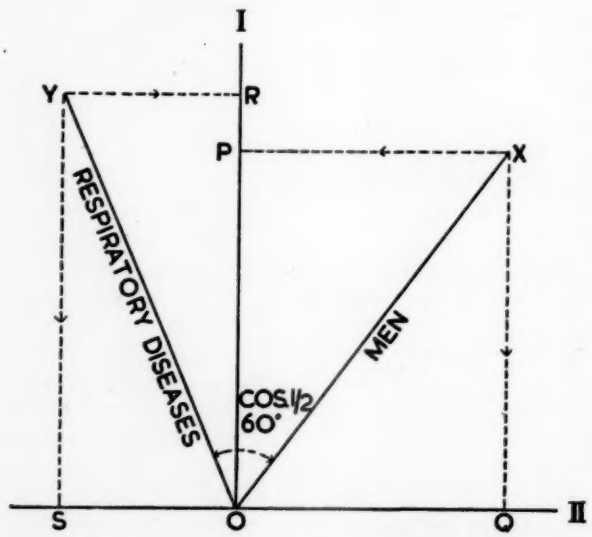


FIG. 3.—The resolution of the attribute vectors on the principal axes.

we can draw a pair of Axes I and II which can be either at right angles or oblique. We may imagine *OX* split along its length; one part projected or casting its shadow on to I and the other part on to II. Let the shadow on I be of length *OP*, and that on II of length *OQ*. These lengths are called the resolutions, projections, loadings, or saturations of *OX* on I and II respectively.

Now let us imagine another vector *OY* placed in the diagram and let it form an angle with *OX* whose cosine is equal to the correlation between *OX* and *OY*. Now *OY* can also have shadows on I and II. Let these be *OR* and *OS*. We can now think of I as containing the parts of *OX* and *OY* which they share in common, that is we can think of *OP* and *OR* as measures of the amount of the influence which the vectors have in common, or as measures of the strength of classifying them together. Thus suppose *OX* and *OY* form the vector system representing the distributions "men" and "respiratory disease" respectively. The angle between the two vectors is fixed by the cosine convention. We can now imagine that the influence which is common to "men" and and "respiratory disease" is given on Axis I and that the contributions towards the influence are represented by the length *OP* for the men, and *OR* for the respiratory disease. Similar considerations apply to the projections on Axis II.

So far I has been placed arbitrarily between *OX* and *OY*; if we imagine I placed in various positions between *OX* and *OY* it is clear that the lengths of *OP* and *OR* will vary as I swings from one side to the other. One good choice of position is that in which the sum of squares of the loadings or shadows of *OX* and *OY* on I are made as large as possible. This is achieved when we minimize the sum of squares of the distances *YR* and *PX* of the points *Y* and *X* from *OR*. In the diagram this position is attained when I lies mid-way between *OX* and *OY*. In the jargon of factor analysis we "maximize" the sum of squares of the loadings or saturations of the vectors on the first or principal axis.

When there are three dimensions there will be three principal axes, I, II, and III; we then arrange for the sums of squares of the loadings on axes I and II to be as large as possible. When there are four dimensions and four axes we maximize the sums of squares on I, II ,and III; and so on for as many axes as necessary.

When there are many vectors the problem has to be solved in multidimensional space. The method of doing this is by finding the "latent roots and

vectors" of the table of correlations, which can be done on an electronic calculator. After adjustment, these calculations provide the loadings of all the attributes on the first, second, third, etc. principal axes.

EXTRACTING THE LOADINGS ON THE PRINCIPAL AXES.—From the table of correlations (like Table VII) we have to obtain a table of loadings of each attribute on each principal axis. As we have seen in the previous paragraph these loadings are nothing more than the shadows or projections of the vectors representing correlations on axes like I and II.

To compute the loadings for Table VII we first note that there are two determinables and five attributes. The number of axes is "attributes minus determinables plus 1", which in this case is $5 - 2 + 1 = 4$ axes. In Table VII there are five rows and five columns, one for each attribute. We write a column with five numbers—any numbers will do; the totals of the rows are a good guide. The first row of the table is post-multiplied by the new column, element by element, and is summed to form the top entry of a second new column; similarly with the second and other rows of the table to complete the second new column of five numbers. If the first and second new columns are not in proportion to each other we use the second column for a new trial and repeat the process. We go on performing the repetition for as many times as are necessary to make the two columns proportional. When we are satisfied, let us call the final columns u and v; u is a "latent vector"—a column of numbers. We next reduce the items in u so as to make their sum of squares equal to a number called the "latent root" which is the largest item in v. When this is done we have the loadings on the First Principal Axis I.

In theory we next construct a 5×5 Table; this is done by writing, outside the Table along the top and the left-hand edges, the numbers we have just found for the first principal axis; the body of the Table is then filled in by forming all the products in order. This Table is subtracted element by element from Table VII and we get the "first factor residuals". This Table is just like Table VII; it is submitted to the same procedure of the u and v columns which eventually lead to the loadings on the Second Principal Axis II. This procedure goes on for as many axes as are needed—in the present case four.

In practice none of these calculations appeared on paper; the correlations from the frequency Table VI were punched on cards in binary notation and subjected to a "programme" in the electronic calculating machine at the National Physical Laboratory. The loadings on the first four principal axes for Table VII were done in this way and appear in Table IX.

TABLE IX

LOADING ON FOUR PRINCIPAL AXES

Attributes	Axis I	Axis II	Axis III	Axis IV	Sum of Squares
Men	+ ·37	+ ·77	+ ·51	0	1
Women ..	+ ·93	− ·31	− ·21	0	1
No Disease ..	+ ·37	− ·32	+ ·21	+ ·84	1
Respiratory ..	+ ·56	+ ·68	− ·45	+ ·13	1
Hypertensive ..	+ ·74	− ·35	+ ·23	− ·52	1
Sum of Squares	2·00 1st Latent Root and Determinables	1·38 2nd Latent Root	+0·61 3rd Latent Root	1·00 4th Latent Root	5 Attributes

The first latent root is equal to the number of determinables—2·00 in this case shown as the sum of squares under Axis I. The sum of squares for each line across is one as shown in the last column, making a total 5, one for each attribute. The loadings on Axis I are proportional to the roots of the frequencies, *i.e.* to the diagonal entries in Table VI.

It is possible to apply a probability test to see if any of the columns, especially the last ones, could have arisen by chance.

THE SIGN PATTERN.—Table IX also shows a trend (which is more apparent in Table XI) for the plus and minus signs in each column to fall into a hierarchical order; thus the first column has all plus signs, the second column is roughly half plus and half minus, the third again sub-divides the halves in column two into plus and minus divisions; column four has two lines with zero entries and is not characteristic; the Table is too small, and because it is artificial may not show the phenomenon clearly at all.

Burt (1954) has pointed out that, in some investigations, the material itself has the intrinsic property of having signs which fall into a true hierarchical order and which can be used as the basis of a classification. In a perfect hierarchy, with eight attributes, we should get the ideal scheme (Table X, overleaf)

In the present investigation we extracted the loadings on three principal axes. We did not go any further because out of the total 42 for the sum of squares for 42 attributes, Axis I absorbed 9·00—corresponding with the number of determinables—Axis II absorbed 2·88, and Axis III absorbed1·99, a total of 13·87 for the three axes. This left 28·13 squares to be divided over 39 axes—less than one square per axis. As each axis had to be split over 42 items, it was considered that the enormous labour and time on the electronic calculator of extracting

TABLE X

HIERARCHICAL FACTOR SIGN SCHEME FOR EIGHT ITEMS

Attribute	Axis I	Axis II	Axis III
1	+	−	+ 3
2	+	− 1	+
3	+	−	− 4
4	+	−	−
5	+ 0	+	− 5
6	+	+ 2	−
7	+	+	+ 6
8	+	+	+
	Group 0 8 items + + + + + + + + As these are the roots of the frequencies they are not of further interest	Group 1 4 items − − − − Group 2 4 items + + + +	Group 3 2 items + + Group 4 2 items − − Group 5 2 items − − Group 6 2 items + +

By a suitable conversion the minus entries can be turned into groups with positive signs, as in the lower part of the Table, and if need be these groups can be regarded as factors, or influences, or classifications as we please.

further axes was not worthwhile. We could have applied a probability test but even this on such a large table was a formidable undertaking, and we decided not to do it.

It is interesting that this material did indeed form a hierarchical pattern, which is set out in Table XI.

ROTATING THE AXES.—Thurston (1947) recommends a further step. Instead of leaving the loadings on the principal axes as in Table XI, he rotates the axes round the origin *O* into various positions of obliquity. The new positions must have the property of forming a "simple pattern" in which the sums of squares of some loadings on certain axes are a minimum and on others a maximum. There are two methods of doing this, both dependent on geometrical manipulation. We submitted our data to both of these processes and obtained three factors from each method. As the manipulation often involved a choice of positions for the axes, and is to some extent arbitrary, we are disinclined to place much reliance on these methods, and have not included any results based on them.

THE PRINCIPAL AXIS SOLUTION.—In the principal axis solution, the fixing of the axes is unique in the sense that it is determined mathematically. With a Table of as many as 42 attributes, the extraction of the principal axes from the correlation table by desk methods would have required such an enormous amount of computational labour as to be almost prohibitive. We were fortunate in having the use of the electronic calculator at the National Physical Laboratory. Even with the great resources of this machine, the extraction of the latent root and vector for the third principal axis required over 4 hours running time. Without the machine we should have had to be content with a less accurate method.

TABLE XI

LOADINGS ON PRINCIPAL AXES

Attribute Reference Number	Attribute	Loadings on Axis II	Loadings on Axis III
		Group 1	*Group 3*
13	Systolic blood pressure 130–159	− 385	+ 217
39	3rd Disability: None	− 215	+ 202
24	1st Disability: None	− 301	+ 200
34	2nd Disability: None	− 353	+ 184
12	Haemoglobin 100–129	− 009	+ 173
19	Diastolic blood pressure 60–89	− 383	+ 143
1	Men	− 166	+ 142
41	4th Disability: None	− 096	+ 090
3	Age 60–64	− 110	+ 082
27	1st Disability: Mental, nervous, senses	− 234	+ 060
			Group 4
15	Systolic blood pressure 180–199	− 073	− 255
8	Haemoglobin 30–69	− 043	− 174
14	Systolic blood pressure 160–179	− 317	− 164
31	1st Disability: Respiratory	− 145	− 159
28	1st Disability: Heart, arteries	− 011	− 152
5	Age 70–74	− 085	− 107
20	Diastolic blood pressure 90–99	− 270	− 103
33	1st Disability: Bones, malformations, ill-defined	− 156	− 080
26	1st Disability: Anaemia	− 125	− 078
32	1st Disability: Digestive, genito-urinary	− 149	− 043
9	Haemoglobin 70–79	− 066	− 037
		Group 2	*Group 5*
40	3rd Disability: All	+ 494	− 464
21	Diastolic blood pressure 100–109	+ 040	− 411
42	4th Disability: All	+ 415	− 390
35	2nd Disability: Infective, neoplasms, allergic	+ 402	− 350
29	1st Disability: Benign hypertension	+ 276	− 200
4	Age 65–69	+ 140	− 192
25	1st Disability: Infective, neoplasms, allergic	+ 020	− 132
2	Women	+ 129	− 111
36	2nd Disability: Anaemia, mental, nervous, senses	+ 105	− 092
16	Systolic blood pressure 200–219	+ 212	− 054
22	Diastolic blood pressure 110–119	+ 162	− 052
38	2nd Disability: Respiratory, digestive, genito-urinary	+ 075	− 041
10	Haemoglobin 80–89	+ 033	− 016
			Group 6
30	1st Disability: Malignant hypertension	+ 619	+ 530
23	Diastolic blood pressure 120–159	+ 608	+ 515
7	Age 80–84	+ 085	+ 288
18	Systolic blood pressure 240–299	+ 484	+ 252
17	Systolic blood pressure 220–239	+ 346	+ 183
6	Age 75–79	+ 029	+ 134
37	2nd Disability: Heart, arteries, hypertension	+ 201	+ 070
11	Haemoglobin 90–99	+ 073	+ 033

DISCUSSION ON THE FACTOR ANALYSIS

The purpose of a factor analysis is to try to discover a set of factors whose interpretation might help us to understand the underlying order or grouping of the attributes; possibly to suggest the influences affecting the people in a group, and lead us to the causes of their disabilities; and perhaps to help to suggest remedies and means of prevention. We must, however, be careful to remember the

limitations of our data. Our sample contained only about half the population at risk. The set of attributes chosen for analysis is to some extent arbitrary in that attributes can be included or excluded at will; so that some attributes, perhaps those of the greatest importance, may be overlooked altogether. These variations lead to differences in the make-up of the factors and to variations, especially of emphasis, in the interpretations.

With these reservations in mind, the four groups of loadings on Principal Axis III in Table XI lead us to believe that our sample can be classified according to the four groups of attributes set out there. Each group is arranged in order of magnitude so as to put those with the highest loadings at the top. The first few attributes in each group are thus closely associated with the factor and with each other, and can form the basis of a group classification of the population. In each group it is possible that there might be a common influence operating either as cause or effect. For convenience, let the groups be re-named A, B, C, and D; Table XI then gives us the following:

Groups from Factor Analysis

MAIN ATTRIBUTES IN ORDER OF IMPORTANCE

Group A.—Healthiness:

Systolic blood pressure 130–159;
Absence of disease;
Haemoglobin 100–129;
Diastolic blood pressure 60–89;
Men.

Group B.—Rising Systolic Pressure, Anaemia, and Chest Diseases:

Systolic blood pressure 160–199;
Haemoglobin 30–69;
Respiratory disease;
Heart and arterial disease.

Group C.—Multiple Disabilities, Rising Diastolic Pressure, and Obesity:

Three or four diseases;
Diastolic blood pressure 100–109;
Infective, neoplastic, and allergic diseases (especially obesity);
Age 65–69.

Group D.—Malignant Hypertension:

Malignant hypertension;
Diastolic blood pressure, 120–159;
Age 80–84;
Systolic pressure 220–299.

Relationship to Blood Pressure.—The four groups are closely related to blood pressure. Persons with systolic blood pressure 130–159 tend to be healthy and to have high haemoglobin; those with systolic blood pressure 160–199 tend to have low haemoglobin and to suffer from respiratory, heart, and arterial diseases. Those with diastolic pressure 100–109 tend to have multiple disabilities and obesity; those with diastolic pressure 120–159 tend to have malignant hypertension. Thus, as blood pressure rises, people so afflicted tend to have more and severer disabilities.

Deficiency Diseases.—In Group B (rising systolic pressure, anaemia, and chest diseases) there may be diet deficiencies, since deficiencies of iron and possibly of other trace elements like boron, manganese, molybdenum, and copper, as well as deficiencies of protein, render animals and plants susceptible not only to anaemia and deficiency diseases but also to infections.

It is tempting to wonder if rising blood pressure is a deficiency disease. In Group C, persons with three or four disabilities tend to have rising diastolic pressures and to be fat. Wilgram, Hartroft, and Best (1954) have suggested that deficiency of choline, a derivative of lecithin found in egg-yolk, may be a possible cause of cardiovascular damage. Most of our obese subjects were women (29 women, 5 men). In working-class households it is common for the women to deny themselves the expensive protein and protective foods, and to live mostly on starchy foods instead, especially bread and butter or margarine and preserves, or bread and fat dripping. In the North they also like tea-cakes, scones, and cakes. They tend to put on weight in the 30s, and become fatter as they grow older; some reach 18 stones in weight.

Corrective and Preventive Measures.—To combat the anaemia and deficiencies in Group B we can bring to the attention of doctors the prevalence of these conditions so that appropriate remedies can be prescribed. By health education we can teach the value and importance of a diet rich in proteins and minerals and encourage good habits in the choice of foodstuffs. The overweight subjects of Group C can be warned about the dangers of obesity and shown how to control their weight.

SIGNIFICANCE OF SYMPTOMS AND SIGNS IN HYPERTENSION.—From the clinical records we made a series of frequency tables showing the association of various symptoms and signs with both systolic and diastolic pressures above and below the average. All the following had a probability of association in random sampling greater than 0·05, and so do not pass the 20 to 1 test of significance:

In both men and women and for both systolic and diastolic pressures: mitral presystolic murmur, mitral systolic murmur, mitral diastolic murmur, split first sound, aortic systolic murmur, aortic diastolic murmur, accentuated aortic second sound, pulmonary systolic murmur, accentuated pulmonary second sound, enlarged right ventricle, pendulum rhythm, tic-tac rhythm, gallop rhythm, tremor, headache, tinnitus, vertigo, and ataxia.

AGE AND BLOOD PRESSURE.—With age the systolic blood pressure shows a significant rise in women but not in men; the rise in diastolic blood pressure is not significant in either. There is a large scatter

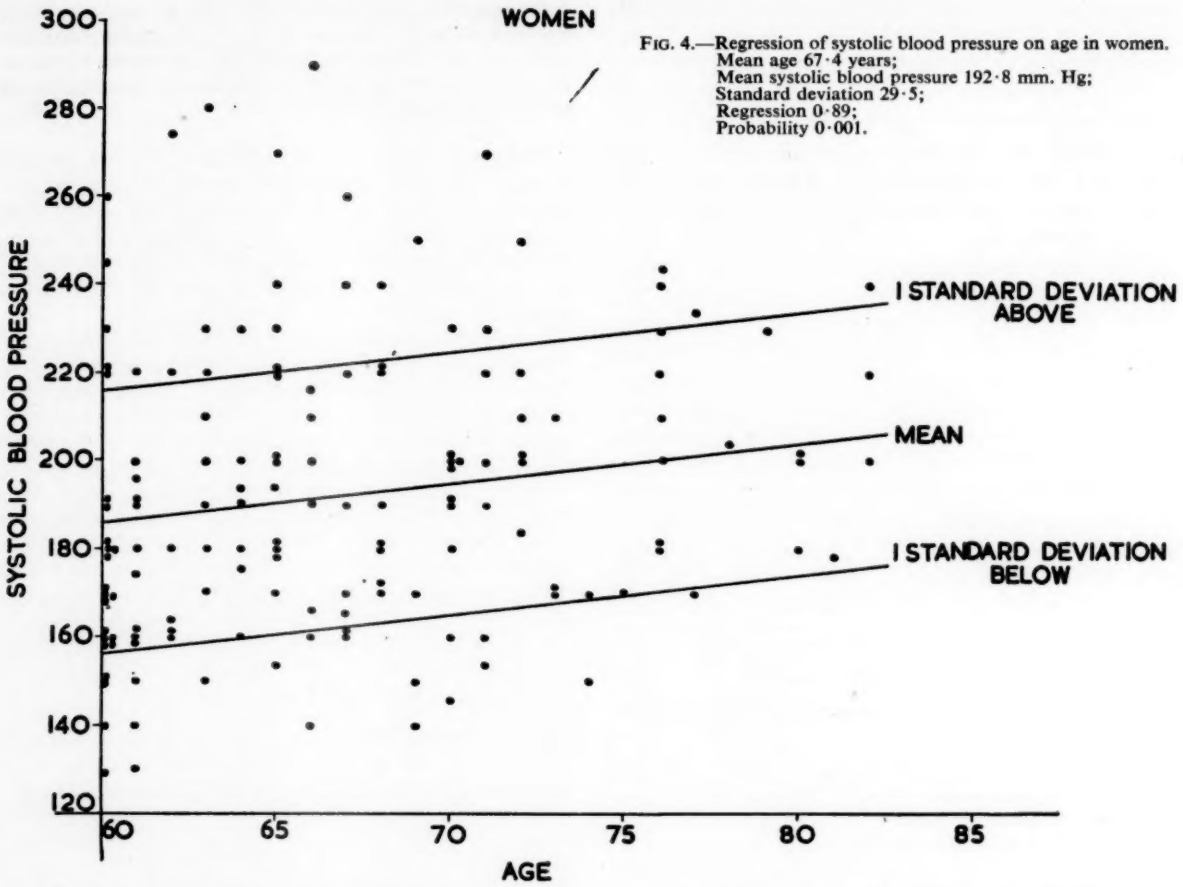


FIG. 4.—Regression of systolic blood pressure on age in women.
Mean age 67·4 years;
Mean systolic blood pressure 192·8 mm. Hg;
Standard deviation 29·5;
Regression 0·89;
Probability 0·001.

in pressure on each side of the mean at every age. Fig. 4 shows the regression of blood pressure on age in women. One standard deviation above or below the mean gives the point where 340 persons out of 1,000 would fall by random selection so that 680 would fall between the two standard deviation lines as shown.

ENLARGED LEFT VENTRICLE.—With enlarged left ventricle the association with both systolic and diastolic blood pressure is close. The probabilities were:

men systolic 0·05,
men diastolic 0·01,
women systolic 0·00001,
women diastolic 0·004.

UNFOLDED AORTA.—With unfolded aorta the association was close with systolic blood pressure but not with diastolic. The probabilities were:

men systolic 0·01,
men diastolic 0·10,
women systolic 0·01,
women diastolic 0·37.

Droller, Pemberton, and Roseman (1952) found no significant association between blood pressure and vertigo, tinnitus, angina of effort, clinically detectable arteriosclerosis, radiological size of the heart, and the subject's well-being and activity.

SUMMARY AND CONCLUSIONS

(1) We were able to carry out a thorough physical examination of 238 men and women aged 60 and over, out of 550 living in Tunstall and Silksworth, where the estimated combined population is 7,500.

(2) The observations were coded, punched on cards, and sorted; a frequency table was prepared and from this a factor analysis was made. In addition, correlations were ascertained, and probability tests by analysis of variance and χ^2 were carried out on certain parts of the data.

(3) The factor analysis gave four groups:

Group A.—Healthiness:
Systolic blood pressure 130–159;
No disease;
Haemoglobin 100–129.

Group B.—Rising Systolic Pressure, Anaemia, Respiratory and Circulatory Diseases:

Systolic blood pressure 160–199;
Haemoglobin 30–69;
Respiratory, heart, and arterial diseases.

Group C.—Multiple Disabilities, Rising Diastolic Pressure, and Obesity:

Three or four diseases;
Diastolic blood pressure 100–109;
Obesity.

Group D.—Malignant Hypertension:

Diastolic blood pressure 120–159;
Systolic blood pressure 220–299.

(4) Groups A and B are related to systolic blood pressure; Groups C and D to diastolic blood pressure.

(5) It is possible that diet deficiencies may be the cause of some of the disabilities in Groups B and C.

(6) Correction and prevention can be obtained by prescription of remedies for deficiencies, by education in dietetics, and by instruction in weight control.

(7) Systolic blood pressure rises significantly with age in women. Many symptoms and signs traditionally associated with hypertension proved not to be statistically significant. When diagnosed by screening, the association of enlarged left ventricle and unfolded aorta with systolic and diastolic blood pressures is close.

We acknowledge with grateful thanks the assistance we have received from many people during the course of this research. We would specially like to mention our gratitude for the facilities placed at our disposal in the department of geriatrics at the Sunderland General Hospital, and to Drs. E. W. Williams, H. S. Stott, and W. Lloyd, and the sister, nurses, and laboratory technicians for their assistance with the medical examinations. Also to the Director of the National Physical Laboratory for permission to use the electronic calculator, and Dr. Goodwin, Mr. Vickers, and Miss Rigg of the Mathematics Division for assistance in sorting and in the extraction of the latent roots and vectors.

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VITAL STATISTICS OF THE COUNTY OF LONDON IN THE YEARS 1901 TO 1951

BY

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The growth and development of London during the past 50 years has been influenced by three major factors. The first factor to operate, and one which still functions, is the continuous growth of London as a commercial centre and the consequent pressure exerted on living conditions by the creation of new offices, shops, warehouses, and factories, especially in the central areas. Secondly, the first world war brought about a social revolution, and perhaps one of its most important consequences was the large increase in opportunities for female labour in office and factory. Thirdly, during the second world war, thousands of houses in London were destroyed, and a very severe housing shortage ensued. With the publication of the 1951 Census for London it is possible to see how these factors have influenced the vital statistical trends of the different London boroughs during the last half century.

Population

At the beginning of the 20th century London had already overflowed its boundaries in the east and the north-east where other towns had become contiguous with London. Some boroughs were not completely built up and in the north-west and south some land was still used for agriculture and wild life still persisted. Since horse vehicles and steam trains were the only methods of transport, most members of the population were compelled to live fairly close to their places of employment. The development of electricity for traction in the first decade of the 20th century led to the electrification of some suburban train services, the district and metropolitan railways, and the construction of the underground system of "tubes". Further electrification and the introduction of the motor bus greatly facilitated travelling; the centre of London became more commercialized and surrounding country was engulfed for dormitories.

Table I shows the population enumerated at the last five censuses and the changes that have occurred in the distribution of the population during the past 50 years. The central cluster of boroughs (including the City, Finsbury, Holborn, St. Marylebone, Southwark, and Westminster) showed a continuous decline in population during the first 30 years and much house property was converted to commercial uses. On the other hand the outer boroughs, and those incompletely built-up boroughs in 1901 (Fulham, Hammersmith, Hampstead, Lewisham, Wandsworth, and Woolwich) showed consistent rises. The changes in the distribution of population between 1931 and 1951, particularly the large falls in population in the adjacent boroughs of Bermondsey, Bethnal Green, Finsbury, Poplar, Shoreditch, Southwark, and Stepney, reflect the damage done during the war. In all these districts the 1951 population was approximately only half the 1931 level. Only in Hampstead, Lewisham, and Woolwich did the population of 1951 exceed that of 1931, and the gains here were very slight.

The best residential districts in 1901 were situated in Hampstead and Kensington, and the large number of female domestic staff employed accounts for the very low proportion (39 per cent.) of males in the population of these boroughs. Despite the disappearance of large domestic staffs, the great preponderance of women in these areas persisted until 1951. The occupational section of the 1951 Census is not yet published, but it is probable that the excess female population is due to women in personal service in the hotels and boarding houses to which the large private houses were converted in many instances. Similarly the excess of females in some other boroughs—Chelsea, Holborn, Paddington, and St. Marylebone—is probably due to the many hotels which are here situated. The City, Bermondsey,

TABLE I

POPULATIONS OF BOROUGHS

Borough	Populations (thousands)					Intercensus Variations (per cent.)				Percentage of Males in Population				
	1901	1911	1921	1931	1951	1901–11	1911–21	1921–31	1931–51	1901	1911	1921	1931	1951
City	27	20	14	11	5	−27·0	−30·3	−19·8	−51·6	51·4	51·3	49·0	50·7	49·7
Battersea	169	168	168	160	117	− 0·7	− 0·0	− 4·9	−26·6	48·4	48·2	46·9	47·3	46·8
Bermondsey	131	126	119	112	61	− 3·7	− 5·1	− 6·6	−45·6	49·8	50·0	48·8	49·2	49·2
Bethnal Green	130	128	117	108	58	− 1·2	− 8·5	− 7·7	−46·1	48·9	48·9	48·4	48·3	47·3
Camberwell	259	261	267	251	179	0·8	2·2	− 5·9	−28·5	47·4	47·5	47·0	47·2	47·1
Chelsea	74	66	64	59	51	−10·1	− 4·0	− 7·3	−13·7	44·4	42·9	40·5	40·6	41·6
Deptford	110	109	113	107	75	− 0·8	2·8	− 5·0	−29·4	48·6	48·4	48·1	48·3	47·1
Finsbury	101	88	76	70	35	−13·3	−13·6	− 8·0	−49·4	49·2	48·5	47·6	48·1	47·8
Fulham	137	153	158	151	122	11·7	3·0	− 4·4	−19·1	47·1	47·4	46·4	46·7	46·4
Greenwich	96	96	100	101	90	0·2	4·7	0·5	−11·0	49·7	49·2	48·6	49·1	48·7
Hackney	219	223	222	215	171	1·6	− 0·2	− 3·1	−20·4	46·1	46·3	45·9	46·3	47·2
Hammersmith	112	122	130	136	119	8·3	7·2	4·0	−11·9	47·0	47·8	48·0	48·0	47·9
Hampstead	82	85	86	89	95	4·3	0·8	3·2	7·0	38·7	38·4	38·6	39·3	41·8
Holborn	59	49	43	39	25	−16·9	−12·5	−10·0	−36·2	49·6	48·8	48·0	46·8	45·4
Islington	335	327	331	322	236	− 2·3	1·0	− 2·7	−26·8	47·6	47·6	46·8	47·2	47·2
Kensington	177	172	176	181	168	− 2·4	2·1	2·7	− 6·9	39·1	38·6	38·6	39·3	42·9
Lambeth	302	298	303	296	230	− 1·3	1·6	− 2·2	−22·3	47·3	47·7	46·7	47·2	47·6
Lewisham	127	161	174	220	228	26·1	8·3	26·3	3·5	44·5	45·3	45·4	46·3	46·9
Paddington	144	143	144	145	125	− 1·0	1·2	0·5	−13·4	42·8	42·4	41·9	42·4	45·7
Poplar	169	162	163	155	74	− 3·8	0·1	− 4·6	−52·6	50·2	49·8	49·5	49·4	49·2
St. Marylebone	133	118	104	98	76	−11·4	−11·8	− 6·3	−22·3	43·4	42·0	39·4	39·7	41·7
St. Pancras	235	218	211	198	138	− 7·2	− 3·2	− 6·3	−30·2	48·6	48·9	47·1	47·6	47·8
Shoreditch	119	111	104	97	45	− 6·1	− 6·4	− 6·9	−53·8	49·0	49·2	48·3	48·7	48·2
Southwark	206	192	184	172	97	− 6·9	− 3·9	− 6·9	−43·4	50·1	49·8	48·6	49·3	48·7
Stepney	299	280	250	225	99	− 6·3	−10·8	− 9·8	−56·1	50·4	50·1	49·1	48·9	49·4
Stoke Newington	51	51	52	51	49	− 1·1	3·0	− 1·9	− 4·0	44·1	44·7	45·0	45·0	47·6
Wandsworth	232	311	328	353	330	34·3	5·4	7·5	− 6·4	44·7	45·2	44·8	45·2	45·8
Westminster	183	160	142	130	99	−12·4	−11·7	− 8·5	−23·6	46·4	45·8	43·4	43·6	46·4
Woolwich	117	121	140	147	148	3·6	15·7	4·6	0·7	52·3	50·5	49·6	49·8	48·9
London	4,536	4,522	4,485	4,397	3,348	− 0·3	− 0·8	− 2·0	−23·9	47·2	47·0	46·2	46·5	46·8

TABLE II
DENSITY INDICES OF BOROUGHS

Borough	Percentage of the Population living more than 2 to a Room					Persons per Room			
	1901*	1911	1921	1931	1951	1911**	1921	1931	1951
City	10·9	12·3	6·6	5·6	0·8	1·06	0·98	0·91	0·77
Battersea	10·9	13·3	12·4	11·9	2·1	1·09	1·04	0·98	0·81
Bermondsey	19·7	23·4	23·2	21·8	2·7	1·37	1·34	1·25	0·93
Bethnal Green	29·6	33·2	27·8	23·6	2·8	1·56	1·46	1·35	0·92
Camberwell	9·6	13·5	12·8	10·4	1·6	1·08	1·04	0·96	0·80
Chelsea	14·4	14·9	13·7	10·2	1·9	1·04	0·85	0·79	0·75
Deptford	9·1	12·2	12·8	11·0	1·7	1·06	1·05	0·98	0·81
Finsbury	35·2	39·8	34·0	29·4	3·4	1·61	1·50	1·42	0·96
Fulham	10·9	14·6	13·1	10·5	2·7	1·09	1·05	0·97	0·84
Greenwich	8·3	12·1	13·8	11·1	1·9	1·05	1·01	0·94	0·83
Hackney	10·2	12·4	11·5	9·6	1·5	1·08	1·06	1·00	0·85
Hammersmith	11·8	14·2	13·8	10·2	3·9	1·05	1·04	1·01	0·90
Hampstead	6·4	7·1	6·5	4·1	2·7	0·86	0·71	0·70	0·80
Holborn	25·0	25·6	19·8	17·1	2·3	1·30	1·12	1·07	0·92
Islington	17·0	20·0	19·4	16·0	3·8	1·22	1·18	1·12	0·92
Kensington	14·8	17·1	16·7	12·6	4·6	1·04	0·81	0·80	0·82
Lambeth	12·2	13·6	12·7	10·6	2·1	1·05	1·02	0·97	0·83
Lewisham	2·7	3·9	4·7	4·1	1·4	0·83	0·80	0·81	0·77
Paddington	13·6	16·2	15·4	12·3	5·2	1·10	0·93	0·90	0·88
Poplar	16·4	20·6	21·2	20·1	3·5	1·33	1·31	1·25	0·93
St. Marylebone	21·1	20·7	17·9	13·1	2·2	1·14	0·88	0·81	0·76
St. Pancras	24·0	25·5	22·4	17·6	4·4	1·32	1·22	1·13	0·93
Shoreditch	30·0	36·6	32·0	29·1	3·5	1·61	1·54	1·45	0·98
Southwark	22·4	25·8	23·5	21·6	2·9	1·43	1·36	1·28	0·93
Stepney	33·2	34·9	29·0	23·6	3·4	1·56	1·45	1·33	0·94
Stoke Newington	5·5	8·8	8·1	6·6	1·7	0·95	0·92	0·90	0·86
Wandsworth	4·5	6·3	6·8	5·2	1·3	0·89	0·84	0·79	0·75
Westminster	13·0	12·9	10·1	7·1	2·3	1·06	0·81	0·76	0·76
Woolwich	6·6	6·3	7·8	5·7	1·3	0·98	0·97	0·88	0·73
London	16·0	17·8	16·1	13·1	2·5	1·14	1·05	0·98	0·83

* In this year the index is of persons living more than 2 to a room *in tenements of less than 5 rooms* as a percentage of the total population, and consequently the value is slightly lower than it would have been on the basis of subsequent years.

** For 1911 the rate was based on families living in 1–9 rooms and is therefore slightly more than a rate based on all private families as in the other years.

Bethnal Green, Greenwich, Poplar, Shoreditch, Southwark, Stepney, and Woolwich had high male ratios in each census year.

DENSITY

Two measures of density are given in Table II. The most striking feature is the very great decrease in overcrowding that was accomplished by 1951. Between 1911 and 1931 a small improvement had taken place in the overcrowding indices but no large changes occurred in the relative positions of the boroughs; the boroughs with the most overcrowding in 1911 still had the most in 1931. In 1951, in most boroughs, the proportion living more than two to a room had become only a fraction of the corresponding proportion in 1931. The lowest indices of overcrowding were recorded at each Census for the boroughs of Hampstead, Lewisham, Stoke Newington, Wandsworth, and Woolwich.

FOREIGN-BORN POPULATION

For the first 30 years of the 20th century the foreign-born population of London remained almost constant in proportion (some 3 per cent.). In 1951 the proportion rose to 5 per cent. and became more widely dispersed through London (Table III, opposite).

Thus, early in the century, there were two distinct colonies. One was in the Soho district, partly in Holborn and partly in Westminster, and was mainly Italian, French, and German. The other was in Stepney and consisted mainly of persons born in Russia, or Russian Poland, who formed about 80 per cent. of the foreign-born population in Stepney. By 1951 the Russians and Poles had overflowed into the neighbouring boroughs and formed two-thirds of the large foreign-born population of Hackney. Political refugees from Poland and Germany formed a large proportion of the increase in the foreign-born population recorded at the 1951 Census. Thus, Germans and Poles form the largest groups of foreign-born persons in the adjacent boroughs of Hampstead, Kensington, Paddington, and St. Marylebone. In 1951 the absolute number of foreign-born persons in these areas taken together was four times the figure of 1901, and formed 34·5 per cent. of the total foreign-born population of London compared with 11·3 per cent. in 1901. In Hampstead in 1951 one person in six was of non-British birth.

TABLE III

NUMBER OF FOREIGN-BORN PERSONS ENUMERATED AT EACH CENSUS AND THEIR PERCENTAGE OF TOTAL POPULATION IN EACH AREA

Borough	1901		1911		1921		1931		1951	
	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.	No.	Per cent.
City	1,243	4·6	1,049	5·3	738	5·4	567	5·2	197	3·7
Battersea	952	0·6	1,129	0·7	1,291	0·8	1,214	0·8	2,689	2·3
Bermondsey ..	1,133	0·9	983	0·8	897	0·8	804	0·7	818	1·3
Bethnal Green ..	4,634	3·6	7,791	6·1	6,864	5·9	5,356	5·0	1,951	3·3
Camberwell ..	1,494	0·6	1,723	0·7	1,920	0·7	1,828	0·7	3,509	2·0
Chelsea	1,263	1·7	1,553	2·3	1,760	2·8	1,551	2·6	4,033	7·9
Deptford	742	0·7	577	0·5	588	0·5	620	0·6	1,083	1·4
Finsbury	2,467	2·4	2,442	2·8	1,966	2·6	1,564	2·2	1,124	3·2
Fulham	1,778	1·3	2,273	1·5	2,343	1·5	2,195	1·5	4,916	4·0
Greenwich	790	0·8	765	0·8	783	0·8	839	0·8	1,387	1·5
Hackney	3,201	1·5	4,788	2·2	8,273	3·7	10,291	4·8	12,639	7·4
Hammersmith ..	1,568	1·4	3,152	2·6	2,870	2·2	2,430	1·8	4,817	4·0
Hampstead ..	2,269	2·8	3,219	3·8	4,619	5·4	5,691	6·4	15,650	16·5
Holborn	5,706	9·6	5,223	10·6	4,652	10·4	3,521	9·1	2,094	8·4
Islington	4,300	1·3	5,076	1·6	5,120	1·5	5,050	1·6	7,502	3·2
Kensington.. ..	4,669	2·6	6,534	3·8	8,469	4·8	8,214	4·5	19,871	11·8
Lambeth	3,506	1·2	5,192	1·7	5,506	1·8	4,738	1·6	7,517	3·3
Lewisham	1,082	0·8	1,264	0·8	1,536	0·9	1,704	0·8	3,393	1·5
Paddington ..	2,822	2·0	4,523	3·2	5,344	3·7	5,298	3·7	12,741	10·2
Poplar	2,104	1·2	1,966	1·2	2,517	1·5	1,666	1·1	1,320	1·8
St. Marylebone ..	5,560	4·2	6,274	5·3	5,998	5·8	5,179	5·3	8,872	11·7
St. Pancras ..	8,156	3·5	10,476	4·8	9,066	4·3	7,267	3·7	7,899	5·7
Shoreditch	2,625	2·2	2,018	1·8	1,745	1·7	1,556	1·6	847	1·9
Southwark	1,593	0·8	1,766	0·9	1,744	0·9	1,352	0·8	1,751	1·8
Stepney	54,310	18·2	53,060	19·0	41,017	16·4	30,083	13·4	8,503	8·6
Stoke Newington	1,073	2·1	1,563	3·1	2,061	4·0	2,712	5·3	3,883	7·9
Wandsworth ..	2,065	0·9	3,090	1·0	4,261	1·3	9,455	2·7	13,567	4·1
Westminster ..	11,831	6·5	13,047	8·1	12,318	8·7	9,229	7·1	9,401	9·5
Woolwich	441	0·4	612	0·5	816	0·6	902	0·6	1,811	1·2
London ..	135,377	3·0	153,128	3·4	147,082	3·3	132,876	3·0	165,785	5·0

TABLE IIIA

FOREIGN-BORN RESIDENTS BY NATIONALITY

Country of Origin	Percentage of all Foreign-born in London				
	1901	1911	1921	1931	1951
Russia } ..	39·5	41·2	23·8	15·4	11·7
Poland } ..			21·1	22·5	23·9
Germany ..	20·3	17·8	6·2	7·4	13·2
France	8·3	9·0	9·7	7·9	5·9
Italy	8·0	7·6	8·0	8·1	7·0
Austria	4·6	5·3	1·1	1·5	7·0
Others	19·3	19·1	30·1	37·2	31·3
Total	100	100	100	100	100

DEATH RATES

Before the adoption in 1911 of the method of transference of deaths to place of usual residence, the death rates in the London boroughs were affected by the number of hospitals and institutions within their boundaries, deaths occurring in such institutions being credited to the borough in which the institution was situated. The study of mortality has therefore been limited to the years 1911 onwards. Some method of standardization is necessary for comparing the London boroughs owing to the varying sex and age constitutions of the populations. For this purpose the direct method of standardization has been used, with the census population of London in 1931 as the standard population. The standardized death rates calculated on this basis are shown in Table IV, which shows that considerable variation in the death rate between London boroughs was present in each period. The range between the boroughs decreased slightly, however; the ratio of the highest to the lowest was 1·8 in 1911–13 and 1·4 in 1950–52. The boroughs of Hampstead, Lewisham,

TABLE IV

STANDARDIZED DEATH RATES

Borough	1911–13	1920–22	1930–32	1950–52
City	19·45	15·42	13·10	8·05
Battersea	15·29	13·56	11·53	8·56
Bermondsey	20·31	16·84	14·04	9·68
Bethnal Green ..	18·86	16·39	13·18	9·69
Camberwell	15·11	13·57	11·45	8·97
Chelsea	14·25	12·09	11·35	8·63
Deptford	16·89	14·21	12·04	9·11
Finsbury	21·90	17·61	14·51	9·77
Fulham	16·20	13·09	11·80	8·69
Greenwich	14·92	13·46	11·17	8·60
Hackney	14·85	13·47	11·70	8·90
Hammersmith ..	15·78	13·65	12·32	9·05
Hampstead	12·35	11·32	10·68	7·80
Holborn	17·71	15·55	13·50	8·86
Islington	16·03	14·27	12·37	9·07
Kensington	14·97	13·58	12·50	8·49
Lambeth	15·51	13·72	11·99	9·05
Lewisham	12·09	11·14	9·93	8·08
Paddington	14·47	12·92	12·61	8·92
Poplar	18·26	15·21	12·74	9·78
St. Marylebone ..	16·29	14·27	12·36	8·67
St. Pancras	16·82	14·77	12·78	9·12
Shoreditch	19·51	16·33	13·22	9·69
Southwark	19·68	16·41	13·99	10·10
Stepney	18·87	16·53	14·35	10·65
Stoke Newington ..	13·98	13·42	11·92	9·16
Wandsworth ..	12·57	11·49	10·27	8·08
Westminster	15·72	13·47	12·12	8·34
Woolwich	14·78	12·42	11·53	8·44
London	15·98	13·91	12·05	8·77

and Wandsworth had the lowest death rates throughout the 40 years, and the boroughs of Bermondsey, Bethnal Green, Finsbury, Shoreditch, Southwark, and Stepney the highest. As is well known, the fall in the death rate has been largely due to the fall in infant and child mortality. This can be illustrated by considering the death rates for the first and last period for London as a whole (Table V):

TABLE V
ADMINISTRATIVE COUNTY OF LONDON
DEATH RATES PER 1,000

Age (yrs)	1911–13		1950–52	
	Males	Females	Males	Females
0–	43·0	36·3	6·4	4·9
5–	2·7	2·6	0·6	0·4
15–	3·2	2·5	0·9	0·6
25–	7·3	5·1	2·2	1·7
45–	24·6	17·2	16·4	8·5
65–	68·2	51·5	61·5	32·5
75+	158·6	135·8	152·7	115·3

It will be noted that between the ages of 45 and 74 the female death rate has decreased faster than the male rate. The death rate for most causes of death in these two age groups show a female advantage. For three large and important groups of causes the rates in 1951 were as shown in Table VI.

TABLE VI
DEATH RATES FOR CERTAIN CAUSES
FOR AGES 45 AND OVER

Cause of Death		Cancer	Heart Diseases	Bronchitis
Age 45–64	Males	4·3	4·4	1·9
	Females ..	2·7	1·8	0·4
Age 65–74	Males	13·5	20·4	8·4
	Females ..	6·6	11·1	2·5

Since the overcrowding indices had been so drastically reduced by 1951 it seems of interest to see whether, as in the past, the indices of living conditions are correlated with the death rate. Excluding the City of London from the calculations, the correlation coefficients between the socio-economic conditions of the boroughs and their standardized deaths rates are as follows:

Correlations		1911–13 *r*	1920–22 *r*	1930–32 *r*	1950–52 *r*
Standardized Death Rate and	Percentage living more than two to a room	0·889	0·922	0·874	0·364
	Persons per Room ..	0·930	0·906	0·799	0·800
	Proportion of population in Social Classes IV and V	—	0·694	0·625	0·900

5 per cent level of significance $r = 0{\cdot}36$, 1 per cent. level $r = 0{\cdot}46$.

The death rate was highly correlated with the percentage of persons living more than two to a room in the first three periods, but the coefficient had only just reached the 5 per cent. level of significance in 1950–52. On the other hand the death rate and persons per room was the same for the last two triennia and only slightly below the earlier figures. The correlation between the death rate and the proportion of the male population in Social Classes IV and V was significant for each of the three triennia for which this index was available and significantly larger in 1950–52 than in the earlier periods.

BIRTH RATE

Heron (1906), from a study of the data relating to the London Boroughs, found a negative correlation between fertility and social status. Some of the indices used were approximate, but Mitra (1937) extended the analysis to include data for the 1931 census, with its more accurate index of social conditions, and substantially confirmed Heron's conclusions. The crude birth rate is influenced by the sex and age constitution of the population, and for this reason the legitimate births per 1,000 married women aged 15–44 have been used in the present study (Table VII).

TABLE VII
LEGITIMATE BIRTHS PER 1,000 MARRIED WOMEN AGED 15–44

Borough	1911–13	1920–22	1930–32	1950–52
City	103	107	66	68
Battersea	193	183	121	99
Bermondsey	250	242	149	116
Bethnal Green ..	250	228	142	108
Camberwell	201	190	118	101
Chelsea	172	168	117	113
Deptford	209	190	123	105
Finsbury	236	232	145	124
Fulham	193	168	110	90
Greenwich	209	188	122	100
Hackney	198	185	117	94
Hammersmith ..	185	168	111	101
Hampstead	142	138	93	102
Holborn	146	134	79	95
Islington	195	189	125	104
Kensington	180	169	117	114
Lambeth	184	174	114	101
Lewisham	166	157	101	96
Paddington	176	157	107	106
Poplar	255	238	151	111
St. Marylebone ..	164	146	99	92
St. Pancras	193	179	115	105
Shoreditch	257	250	156	120
Southwark	229	215	135	112
Stepney	245	221	156	126
Stoke Newington ..	178	163	111	102
Wandsworth ..	168	151	98	92
Westminster	134	122	85	98
Woolwich	176	170	108	100
London	198	183	119	102

The highest birth rates were in the East End boroughs throughout the period, while the lowest were those of Hampstead, Holborn, and Westminster in the first three triennia, and of Fulham, St.

Marylebone, and Wandsworth in 1950–52. A comparison of Table VII with Tables II and III suggests that the birth rate, like the death rate, is correlated with the density indices. The correlations are:

Correlations		1911–13 *r*	1920–22 *r*	1930–32 *r*	1950–52 *r*
Legitimate Birth Rate and	Standardized Death Rate	0·748	0·741	0·588	0·718
	Percentage living more than Two to a Room	0·666	0·750	0·777	0·491
	Persons per Room ..	0·768	0·876	0·825	0·601
	Proportion of population in Social Classes IV and V	—	0·811	0·776	0·670

All the correlations are significant. The indices of density in 1950–52 were not so highly correlated with the birth rate as in former years, and the correlation between birth rate and the proportion in the lowest social classes of the population has also decreased since 1920–22, though the difference between 0·811 and 0·670 is not significant.

Infant Mortality

Hersch (1943) chose infant mortality as the best single numerical index of the degree of civilization of a population. He considered that a decrease in this rate reflected the advances made in hygiene, medicine, chemistry, and public instruction, and that it was the best sign of improved living conditions. When Hersch was writing infant mortality in Europe ranged from 37 in the Netherlands to 180 in Rumania, and even higher rates occurred in Eastern countries.

In England and Wales infant mortality has been used in the past as an index of the socio-economic level of the community, since the highest rates were found to occur where living conditions were worst, and infant mortality varied directly with social class. McKinlay (1928) attempted to standardize for varying social conditions in the London boroughs, but found that such standardization for economic status did not reduce the variability between the boroughs by as much as seemed *a priori* probable. Stocks (1928), using the occupations of the males from the census returns, attempted to find a correction factor which would reflect the social make-up of the London boroughs. This correction, however, only reduced the variability between the infant mortality rates of the London boroughs from 17 to 14 per cent. in 1911–13, and from 15 to 13 per cent. in 1921–25. The findings of McKinlay and Stocks are in agreement, and clearly the economic factor, as measured by their indices, was not very important in determining the level of infant mortality in the various boroughs. During the past 25 years the effect of many variables has been studied: *e.g.* indices of density, proportion of mothers employed, fertility rate, and proportion of males in the professional classes and in the lowest type of labour, etc. These investigations have shown that infant mortality was affected by socio-economic conditions, but since the variables used were themselves inter-related it has not been possible to assess the relative importance of their contributions. In recent years infant mortality has fallen rapidly. In England and Wales the rate was 154 deaths per 1,000 live births in 1900, by 1922, this had been halved, and in 1930 the rate was down to 60. Between 1930 and 1941 the rate fluctuated between 51 and 66, but since 1941 when it was 60 it has fallen steadily to 25·5 in 1954.

In 1950–52 the infant mortality rate for London (24) was below the rate for the whole country (29), and much below the rates for the county boroughs of Durham (40), Lancashire (36), Staffordshire (34), and Yorkshire West Riding (31). Not only was the infant mortality lower in London than in the large industrial towns of the north but the range was smaller; thus the rates in 1950–52 ranged from 19 to 31 in the 28 London boroughs, from 29 to 47 in the seventeen country boroughs of Lancashire, and from 23 to 38 in the eleven county boroughs of Yorkshire West Riding. The low level and relatively small range of infant mortality make the London boroughs unrepresentative of urban areas in general. Use of the 1931 census data showed that, although the infant mortality by social class in London showed the same progression as in the whole of the country, the usual indices of social structure in urban areas were less highly correlated with infant mortality than formerly. It was reasonable to suppose that these correlations would have declined still further during recent years, although the social class differential would still exist. No data, however, existed to test this hypothesis until the publication of the 1951 census for London. The occupational supplement is not yet published and it is not possible to display the infant mortality by social class, but there is no reason to suppose that the relative differences between the social classes has undergone much change. A triennial period centred in the census year was used for the appropriate rates shown in Table VIII, overleaf.

For the County of London as a whole, infant mortality in 1950–52 (24 per 1,000) was less than one-

TABLE VIII
INFANT MORTALITY

Borough	1911–13	1920–22	1930–32	1950–52
City	95	80	58	52
Battersea	107	75	57	24
Bermondsey	134	93	59	21
Bethnal Green ..	122	95	72	27
Camberwell	100	74	58	25
Chelsea	91	67	45	23
Deptford	117	80	60	28
Finsbury	137	83	73	19
Fulham	105	76	61	26
Greenwich	102	68	65	21
Hackney	100	73	55	24
Hammersmith ..	114	75	66	20
Hampstead	72	57	58	23
Holborn	101	73	78	31
Islington	107	77	67	27
Kensington	112	91	81	27
Lambeth	104	74	57	26
Lewisham	84	59	49	24
Paddington	109	80	91	28
Poplar	126	81	65	27
St. Marylebone ..	97	69	72	22
St. Pancras	98	75	66	27
Shoreditch	150	102	73	25
Southwark	122	85	63	22
Stepney	121	86	71	22
Stoke Newington ..	85	67	54	23
Wandsworth ..	96	64	60	22
Westminster	95	70	67	28
Woolwich	84	62	54	24
London	109	75	64	24

quarter of the rate in 1911–13 (109 per 1,000). The largest falls occurred in boroughs where the rate was previously very high, the ratio of the lowest to the highest infant mortality being 2·1 in 1911–13 and 1·6 in 1950–52. The correlations with social indices exhibited by the standardized death rates and the legitimate birth rates are not apparent in these infant mortality rates. The correlations are:

	Correlations	1911–13 r	1920–22 r	1930–32 r	1950–52 r
Infant Mortality and	Percentage Occupied Males in Social Classes IV and V ..	—	0·649	0·132	−0·072
	Persons per Room ..	0·823	0·767	0·300	0·020
	Percentage living more than Two to a Room	0·782	0·812	0·478	0·184

Infant mortality showed a large positive correlation with each of the three indices of social status up to 1920–22. In 1930–32 it has almost ceased to be affected by the measures of social conditions, and only the percentage living more than two to a room was significantly correlated with the rate. In 1950–52 no significant correlation was present.

The general death rate, crude or standardized, has been frequently used as a broad measure of the health of the people, and recently the birth rate has given an indirect measure of the conditions of living since it has shown a steep gradation with social class. The correlations of infant mortality with these rates for the last four census periods are:

	Correlations	1911–13 r	1920–22 r	1930–32 r	1950–52 r
Infant Mortality and	Crude Death Rate ..	0·879	0·785	0·523	−0·275
	Crude Birth Rate ..	0·812	0·702	0·043	−0·275
	Standardized Death Rate	0·853	0·794	0·553	−0·126
	Legitimate Birth Rate for Women aged 15–44 yrs	0·836	0·754	0·089	−0·225

In 1911–13, the correlations were all of the same order, being large and positive. In 1920–22, they were slightly smaller, but still large and significant and approximately equal. In 1930–32, the infant mortality was no longer correlated with the measures of the birth rate; the correlation with the measures of mortality had fallen considerably but was still significant. In 1950–52, no significant correlation existed at all.

From the preceding correlations it might be inferred that infant mortality has now fallen to such a low level that it is no longer any criterion of the social or economic differences between the London boroughs. While this is so for the death rate for the whole of the first year of life, it must be realized that the neonatal mortality is now affecting the correlation more than in former years, since the deaths in the first month of life form a much larger proportion of the infant deaths than formerly. The percentage of deaths at various ages in the first year of life in London, for the period reviewed, are shown in Table IX:

TABLE IX
DEATHS IN THE FIRST YEAR OF LIFE

Age (mths)	1911–13	1920–22	1930–32	1950–52
Under 1 ..	31·7	37·6	39·2	67·4
1–3	19·1	18·8	17·1	13·3
3–6	19·4	18·1	18·4	10·4
6–12	29·8	25·5	25·3	8·9
Total	100	100	100	100

The very different structure of infant mortality in 1950–52 suggested that it would be of interest to examine the relation of infant mortality and socio-economic indices by ages:

Age (mths)	Years	Proportion of Population in Social Classes IV and V *r*	Persons per Room *r*	Percentage living more than two to a Room *r*
Under 1	1920–22	0·177	0·226	0·350
	1930–32	−0·295	−0·184	−0·187
	1950–52	−0·430	−0·389	−0·118
1–3	1920–22	0·499	0·618	0·632
	1930–32	−0·155	0·027	0·116
	1950–52	0·273	0·319	0·322
3–6	1920–22	0·541	0·698	0·691
	1930–32	0·113	0·242	0·476
	1950–52	0·387	0·430	0·328
6–12	1920–22	0·714	0·786	0·802
	1930–32	0·521	0·622	0·749
	1950–52	0·428	0·288	0·248
1–12	1920–22	0·667	0·778	0·788
	1930–32	0·273	0·428	0·620
	1950–52	0·496	0·484	0·414

In the first two triennia the correlations between neonatal mortality and the indices of socio-economic conditions were not significant, but in 1950–52 the percentage of the population in Social Classes IV and V and the number of persons per room showed a significant negative correlation. During the period the fall in the birth rate, from 198 legitimate births per 1,000 married women aged 15–44 in 1911–13 to 102 in 1950–52 for the whole of London, may account for this negative correlation by increasing the relative importance of the first births. Heady, Daly, and Morris (1955) showed that, for mothers aged 25 years and over, neonatal mortality is highest among first-born children and increases with age of mother. It has been shown that the birth rate is correlated with the three indices of general living conditions, *i.e.* the lower the social index the higher the birth rate, and it is known that the age of marriage decreases with social class. In 1920–22, the infant mortality at all later ages, 1–3 months, 3–6 months, 6–12 months, and 1–12 months, was significantly correlated with the three indices of environmental conditions. In 1930–32, the correlations at 1–3 months were all insignificant, at 3–6 months one was significant (the percentage of the population living more than two to a room), and at 6–12 months all three were significant. For the post-neonatal period as a whole (1–12 months), the correlation between infant mortality and the percentage in Social Classes IV and V was insignificant, but the other two correlations were significant. In 1950–52, the infant mortality at 1–3 months was not correlated with any of the indices, at 3–6 months it was significantly correlated with the percentage in Social Classes IV and V and the number of persons per room but not with the other index of overcrowding, while at 6–12 months only the percentage in Social Classes IV and V gave a significant figure. For the post-neonatal period, all three indices were significantly correlated with infant mortality.

This sub-division of infant mortality by ages shows, as expected, that the trend of the neonatal mortality is at least partially responsible for the lack of correlation in 1950–52 between infant mortality and sociol-economic conditions. The post-neonatal rate remains significantly correlated with the three indices, though probably at a lower level than in 1920–22 (the differences between the two periods are not significant, except the percentage living more than two to a room).

The relationship between the social indices and infant mortality may rest partly on the population of the boroughs. Most boroughs conform to the official description of a large town having a population of over 50,000; they were divided arbitrarily into three groups* and the correlations found between the percentage living more than two to a room and infant mortality and neonatal mortality:

Population	No. of Boroughs	Percentage living more than two to a Room (1951) correlated with			
		Neonatal Mortality		Infant Mortality	
		r	*p*	*r*	*p*
120,000 and Over	11	0·47	>0·1	0·89	<0·01
Over 60,000 and Under 120,000	11	−0·12	>0·1	−0·32	>0·1
Under 60,000 ..	6	−0·72	>0·1	−0·19	>0·1

In all three groups the neonatal mortality is insignificantly correlated with the overcrowding index. Infant mortality is significantly correlated with this index in the very large boroughs but not in the other two groups of smaller boroughs. No explanation suggests itself to account for this difference. The infant deaths were rather few in the third group of boroughs (27 and 37 in 1950–52 in the two smallest), but in the second group the smallest borough had 69 deaths. Paucity of deaths cannot be an explanation.

It is of some interest to see whether the relationship between the various indices of socio-economic status have changed during the period:

Correlations		1920–22 *r*	1930–32 *r*	1950–52 *r*
Percentage Occupied Males in Social Classes IV and V and	Persons per Room	0·744	0·739	0·757
	Percentage living more than two to a Room ..	0·658	0·722	0·314
Percentage living more than two to a Room and Persons per Room ..		0·899	0·934	0·617

* The City of London has been omitted from all the correlations in this paper since it has a very small and unusually constituted population.

The large significant correlation between the proportion of occupied males in Social Classes IV and V and the number of persons per room has remained constant over the 30 years. The relationship between the proportion of occupied males in Social Classes IV and V and the percentage living more than two to a room had fallen below the level of significance in 1950–52 although a large significant correlation was found in the other two periods. The correlation between the two indices of density, which was very large in the two earlier triennia, fell significantly in 1950–52 but was itself still significant. The decline in the size of the correlation coefficients with the percentage living more than two to a room is directly attributable to conditions arising from the second world war. The destruction of so many houses led to an acute shortage and the opportunity was taken in the rebuilding programme to re-house Londoners outside the county boundaries. The result of this policy is that the population of London was one million less in 1951 than in 1931 (Table I), a decline of 23·9 per cent. at all ages, and of 30·9 per cent. at ages 0–15 years. The magnitude of the change is shown by the densities for the whole:

Overcrowding Index	1911	1921	1931	1951
Percentage living more than Two to a Room	17·8	16·1	13·1	2·5
Average Persons per Room ..	1·14	1·05	0·98	0·83

Summary

Between 1901 and 1931 the population of the County of London was approximately stationary. Between 1931 and 1951 it fell by over a million, and in seven of the 28 boroughs the population in 1951 was only about half that in 1931. The result of these changes was a great reduction in the proportion of persons living in overcrowded conditions. From 1901 to 1931 the proportion of the population living more than two persons to a room was between 1 in 6 and 1 in 8; by 1951 it had fallen to 1 in 40.

From 1901 to 1931 about 3 per cent. of the population of London was foreign-born; in 1951 the figure was 5 per cent. for the County, and over 10 per cent. in four boroughs: Hampstead (16 per cent.), Kensington (12 per cent.), Paddington (10 per cent.), and St. Marylebone (12 per cent.).

The downward trend of the death rate during the 20th century has been remarkably similar in the London boroughs, so that the boroughs with the highest and lowest rates in 1911–13 were in the same relative position in 1950–52. The fall, has, however, been larger in the boroughs where the rate previously was highest so that the actual and relative range of the death rates between London boroughs in 1950–52 was somewhat smaller than in 1911–13. The general death rate (standardized) is still significantly correlated with the indices of socio-economic status (persons per room, and percentage in Social Classes IV and V).

The birth rate followed a trend similar to that of the death rate, and, generally, the relative level of the birth rate of a borough, high or low, has remained the same throughout the period. With the exception of the percentage of the population living more than two to a room, the correlations between the birth rate and the socio-economic indices have changed very little.

The very large fall in infant mortality in the London boroughs has been relatively greater in those boroughs where the initial level was highest. In 1911–13 and 1920–22, infant mortality was significantly correlated with the percentage of occupied males in Social Classes IV and V and with the two measures of overcrowding. In 1930–32 the correlations between infant mortality and overcrowding were reduced but still significant, while the percentage of occupied males in Social Classes IV and V was not significantly correlated with infant mortality. In 1950–52 the correlations between infant mortality and the three indices were not significant. The lack of correlation in 1950–52 was partly due to the increasing contribution made by neonatal mortality, and partly to the fact that the relationship is no longer demonstrable in the smaller boroughs although it still exists in the largest boroughs. If the neonatal component is excluded, the correlation of infant mortality for the first year of life is significant in 1950–52 for each of the three socio-economic indices. The successful efforts to reduce overcrowding have lessened the sensitivity of this index of socio-economic conditions. In 1911 the percentage of persons living more than two to a room ranged from 3·9 to 39·8 in the London boroughs, while in 1951 the range was only from 1·3 to 4·6; thus this index of overcrowding was no longer a measure of either the vital-statistical or socio-economic differences between the London boroughs in 1950–52.

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STATISTICAL EXPERIMENTATION IN THERAPEUTICS

BY

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The issue I propose to examine is whether, or in what circumstances, the statistical approach to the assessment of curative measures is legitimate. This issue is straightforward, since, unless the mere enumeration of cases is regarded as a statistical procedure, the distinction between the statistical and the non-statistical approach to a problem is absolute; an investigator must inevitably be aware that he is making a clearcut and significant decision when he choses to adopt one method rather than the other.

The conclusion I shall draw is that in therapeutics —and analogously in other branches of inquiry— the method of statistical experimentation is largely invalid. This is not to say that the accumulation of statistical material in medicine, or in other fields, is futile, or that the analysis of such material may not provide valuable guidance for the research worker and essential information for the administrator. It is merely to assert that the statistical experiment is not a satisfactory exploratory tool in the hands of the research worker confronted with a specific problem.

We can, I believe, arrive at this conclusion in two ways. Either we may identify ourselves with the experimentalist, who is seeking from the laboratory or clinic to promote a rational basis for therapeutics, and argue that the use of the statistical method is incompatible with the way of thought which ordinarily governs the activity of the scientist. Or, adopting the viewpoint of the mathematician or logician, we may argue that existing theories of statistical inference are inadequately grounded, and that until this matter is rectified we cannot help but accede to any objection on extra-logical grounds which is made to the use of the statistical techniques promoted by these theories; alternatively,—and this is the writer's own standpoint—we may feel ourselves able to argue in this capacity that a consistent theory is possible but that it does not elucidate the problems with which the physician or experimentalist is ultimately concerned.

We have therefore to consider two approaches to the question side by side; and if objections on general grounds to the statistical method are admissible, we must take it that the confusion which characterizes fundamental discussion of modern mathematical statistics will remain until the mathematical statistician is prepared to throw overboard so much that he abrogates in effect his right to advise on the logical structure of biological or other experimentation; on the other hand, if *logical* objections are valid, we must re-examine the position of statistical inference *vis-à-vis* general considerations in the philosophy of science.

Let us first consider objections which can be directed *on general grounds* against the use of statistical experimentation in medicine. Since, by reason of its simplicity and directness, the idea of a statistical approach to therapeutics very readily leaps to the mind, these have often been stated, and at different epochs. First, under the influence of Laplace, when Pierre Louis put forward his *numerical method* for medicine and pathology. Again, when the influence of Quetelet began to be felt in medicine; and later, in Great Britain, when Karl Pearson reintroduced and extended Quetelet's ideas. This last phase is the most interesting from the methodological point of view, since the prospect of unprecedented advances in prophylaxis and therapeutics disclosed by the contemporaneous rise of bacteriology reinforced the discussion with realistic anticipations. Opposition to Pearson's intrusion into the medical field came chiefly from Sir Almroth Wright as a serologist. The immediate point at issue related to Wright's proposals for anti-typhoid inoculation which Pearson publicly opposed on statistical grounds (Wright, 1904; Pearson, 1904). His objections were overruled, anti-typhoid inoculation as recommended by Wright was used amongst British troops in the first world war from the outset; and as a result of the controversy, Wright published several notable contributions to the analysis of the credentials of the statistical method (Wright, 1912, 1921, 1953). It will be convenient to summarize these contributions—with which the writer is in broad agreement—and refer the reader to the original papers for a fuller discussion.

When considering Wright's argument there are two points to be borne in mind. Firstly, most modern statisticians would be amongst the first to agree with some of his criticisms of the statistical procedures of his time and in particular with his strictures with respect to clinical trials with control and treated groups not strictly comparable. Secondly he was as much concerned, in his polemic, to establish the claims of the laboratory scientist against the Harley Street clinician, as to resist the threatened encroachments of the statistician. We may ignore these aspects of his argument and enumerate his fundamental objections to the statistical method. I use, where possible, his own words:

"It is commonly asserted that 'Science is measurement'—*measurement* being understood to comprise the achieving of numerical results by *enumeration weighing*, and quantitative methods, generally. That is quite erroneous; and consideration will show that it is no particular good to anyone to be able to rehearse statistical or other figures. The essence of science is certainty and not measurement."

Insofar as the statistical method is explicitly concerned to repudiate this last proposition, this is the fundamental antithesis on which Wright bases his case. Any science such as medicine relies in its application on firmly established facts which "like fixed points in a trigonometrical survey supply the foundation for mapping out the rest of the territory". In practical affairs, so-called rational decisions are made with the help of such a mapping, though the crudity which results from a limited number of fixed points being available may well be very great. It is the pretence of the method of statistical experimentation to provide direct answers to ill-defined practical questions and, in doing so, to supersede this procedure, which Wright refers to as *contesseration*. Contesseration is the fundamental inductive process. Its mode of operation is tentative and it is liable to unavoidable error; such error, however, admits of correction as the body of our knowledge grows. We delude ourselves if we suppose that there can be any shorter cut from inadequate to adequate knowledge.

"Whenever medicine has progressed it has progressed by making a new departure suggested by a chance observation or a new departure based upon a crucial laboratory experiment. . . . It has not gone forward by refining upon such discoveries by the aid of cumulative experiments." With the cumulative approach, as the physical and moral difficulties increase the intellectual harvest from the work becomes so patently unremunerative that every reasonable man will cry off from statistical inquiry."

We have only to think of the range of substances whose therapeutic value is explored in modern chemotherapy to appreciate the common sense behind this remark. It is not, however, only the barrrenness of the statistical method which is to be deplored. It is possible that, if taken seriously, it will serve to inhibit the making of that type of observation which does lead to fruitful advances.

The true man of science, according to Wright, has a conception of the complexity of nature which demands that he should wish to progress only from step to step. "The man who lives no life within the brain and has no conception of the complexity of nature . . . is always hankering after . . . the *saltus empiricus*. He tells us . . . that this procedure should, whenever possible, be employed and that it is nothing but simple good common sense to eliminate from consideration intermediate links in a chain of causation." "Your statistician," adds Wright, "aids and abets him."

Finally, we should be suspicious of the validity of the statistical method because it is so very easily employed. ". . . a man who proposes to carry out a crucial experiment should possess some native experimental ingenuity; whereas a man requires absolutely none for carrying out a statistical experiment." If the statistical method were novel this consideration would carry little weight. But in fact it has been advocated and applied for over a century. No important advance has resulted in any biological field.

We have discussed the informal aspect of the problem principally from the standpoint of the scientist. Alternatively, we can briefly consider it from the point of view of the physician. Let us suppose that Treatment *A* can be taken to cure 60 per cent. of patients and Treatment *B* to cure 40 per cent. A statistical trial cannot yield a type of information of higher order than this; its outcome will usually be less clearly defined, both in terms of stated percentages and in terms of what is meant by *cure*. What help does this information give the physician? When he has no knowledge at all of his patients and no collateral physiological knowledge of the mode of action of either of the two treatments, it tells him that it will be better, in a statistical sense, for him to use Treatment *A* rather than Treatment *B*. If he does possess any further knowledge under these two headings—and in any practical situation he certainly will—then he must inevitably ask: "May it not be that by judiciously allocating Treatment *A* and Treatment *B* between different patients I can ensure that all recover, or certainly more than the 60 per cent. corresponding

to the indiscriminate use of the 'better' treatment?" The person who has organized the trial cannot answer this question without going beyond his terms of reference, that is beyond his claim to be a fact-producer and an impartial arbiter. Viewed in this light, "The method of statistical experimentation can be used for testing the value of prophylatic measures but not for testing that of therapeutic measures."* The *statistical* trial can at the most be regarded as an adjuvant to *statistical* medicine.

In the second place the physician may ask the investigator a more sophisticated, but equally cogent question: "If you possess so little knowledge that you have to resort to the statistical method, how can you guarantee that my patients do not differ from the subjects used in your experiment in respects which are relevant to the outcome of treatment but of which you are unaware?" To this again the investigator can reply only with assurances which do not derive their sanction from the statistical information formally yielded by his enquiry.

The strongest statement of the case against the use of the method of the statistical experiment in therapeutics will therefore include the assertions:

(*a*) that the method is not in accord with ordinarily accepted scientific canons.

(*b*) that the consistent use of the method is not in accord with the aims of medical practice as commonly conceived.

Against this case lie arguments of which everyone must be fully aware. The statistical method militates against the making of inferences from an insufficient foundation in fact; it guarantees that experience of untreated cases is placed under review; it compels the systematic classification of observational material; it ensures that an investigator does not overlook cases which do not fit in with a preconceived thesis; and it seeks to present the outcome of experience in a form which is pre-eminently communicable. Does, however, the statistical method in any situation provide the *only* means for guaranteeing the intellectual integrity of the investigator? Can it not moreover perhaps introduce fallacies of its own? We may suspect on general grounds that it does. It is certainly desirable to examine the logical foundations of the method and obtain a more precise measure of these disadvantages.

In this matter Wright does not seem to exaggerate; "If, like an aphasic patient, or the ordinary man endeavouring to express himself in a foreign tongue, [we] have to struggle with a terminology which fails to provide words for quite fundamental notions, it is safe to assume that we are dealing with a science which has attracted to its service only men of mediocre ability. . . . The vocabulary of *Statistical Experimentation* is, as the reader who has any acquaintance with it does not require to be told, distressingly defective."

Wright's biographer remarks that he probably made too little allowance for recent progress in the development of statistical technique (Colebrook, 1954). This, however, is beside the point. Any advance in theoretical statistics can be of sufficiently radical importance to overturn Wright's main thesis, only if it involves a clarification of the disputed vocabulary of the subject. The majority of modern handbooks of statistical methodology contain no clearer definitions of such terms as *random*, *probable*, and *significant* than are implicit in Venn's "Logic of Chance", the first edition of which appeared in 1866. Judged by this criterion, progress has occurred in the mathematical but not in the logical structure of theoretical statistics. Whatever fundamental advance has been achieved does not obtrude into the practical handbooks.

The generalized statistical experiment may be treated formally as a sampling procedure in which balls are drawn haphazard from an urn in order to assess relations between the original proportions of different known types of ball within the urn (Wrighton, 1953). The fundamental problem of statistical inference therefore lies in giving precision to the type of inference which can be made in formal situations of this type. The classical approach to this problem largely persists and presupposes that it is in the following form that the problem is to be attacked. An urn contains unknown proportions of classified types of ball; a number of balls has been withdrawn at random, yielding a sample whose constitution is to be analysed in such a way as to tell us what can be inferred about the original contents of the urn. We may refer to this approach as *retrospective* or *analytical.*

Granted this approach we may adopt one of several procedures. We may perhaps, on the basis of the fixed body of data with which we are presented, attempt to make a statement about the state of mind which should be induced by an observed discrepancy between sample proportions. This we do if we operate a significance test. Again, we may seek to pass *exact* judgement on the true value of a population proportion. This we do if we adopt a point estimation procedure. Again, we may employ

* Wright (1953), p. 203.

a decision test and seek thereby to recommend that a particular course of action should be taken as a result of the sample observation. Lastly, we may seek to erect a scale of "probabilities" related to statements, each of which can be regarded as *a priori* possible, concerning the contents of the urn.

All these procedures have one significant feature in common. In each case we ignore collateral evidence and attempt to pass a judgement with putative practical significance upon evidence provided by a closed body of data. In Wright's phraseology, we ignore the requirement that judgements formed from observational data should be made by contesseration with other information available in the field. Except in special and uninteresting cases this formal neglect of collateral evidence must certainly render any calculus with pretences to the weighing of evidence nugatory in practical application. Can, however, such a calculus be developed, which is even consistent, in the sense that different judges may be drawn by it to identical numerical conclusions in respect of the implications of particular closed bodies of sampling data? On general grounds we cannot reasonably anticipate that the answer to this question can be *yes*; and experience shows that all theories so far elaborated, and however plausible initially, collapse on analysis. The ultimately operative charge against them is that of equivocality. The Bayes-Laplace calculus has so often been refuted, not only because of justifiable reluctance to accept the concept of prior probabilities and the postulate of their equality—these in themselves need afford no deterrent to the mathematician—but also because it is impossible to distribute these equi-probabilities unambiguously even in formal situations. Similarly, the initially intuitive approach to significance testing must be rejected, not merely because of its inherent irrelevance in practice, but also because (as J. Neyman and E. S. Pearson have shown) it is impossible, except in artificial and trivial situations, to assign appropriate regions of rejection unequivocally. We might comment similarly on other forms of retrospective calculus.

What holds for these inverse theories holds also for Wald's theory of Decision Functions, initially advanced to supply a logical foundation for recently elaborated statistical techniques* in the same way as, earlier, Jeffreys put forward a form of the classical inverse calculus to explain techniques associated with the name of R. A. Fisher. It is often said that Wald and Jeffreys stand for ways of thought which are completely antithetical. This seems to me to be a gross misunderstanding. The dividing line between a calculus which aims to provide numerical judgements which may have bearing on the making of a decision and one which actually dictates the decision is so vaguely defined that discussion of objections to Wald's theory must largely rehearse the case against that of Jeffreys. Wald's minimax principle is certainly unambiguous in a way in which Bayes' postulate is not; but it is equally arbitrary. Collateral evidence is allowed for in the requirement that a whole range of assessments of conditional risks must be made. But these assessments are to be in numerical terms and could not be made in any situation conceivable in medical research. Moreover, could they be made, the investigator would be in possession of so much knowledge that the initial undertaking of inquiry at a statistical level would be superfluous.

The only conclusion we can therefore draw from recent studies is that, on purely formal grounds, it is futile to attempt to develop a calculus of judgement or of decision. This is hardly compelling, since the plain man will have reached the same conclusion in the absence of intellectual effort. We are left with a line of development opened up by J. Neyman. To Neyman is due the notion of interval estimation by direct probabilities. This approach holds out more promise of success, since it suggests that we can hope to generate a class of statistical facts which can be viewed on almost equal terms, and therefore in effect contesseratively, with related non-statistical facts. Here we have a complete break with tradition. The retrospective approach is seemingly abandoned and the unattainable goal of judgement is ignored. But Neyman seems to have regarded his notion as purely mathematical, and has attempted to use it, not to throw light upon the nature of statistical reasoning, but to develop a retrospective calculus which is almost conventional. R. A. Fisher pointed out very early that the resulting *theory of confidence intervals* falls down because it does not admit of consistent development and he himself advanced contemporaneously the theory of *fiducial probability*, which uses the same basic notion in an inverse calculus applicable, however, only in a limited number of ideal situations. These theories do no more than provide further examples of the futility of the analytical approach.

I have put forward elsewhere (Wrighton, 1953) what I believe to be the correct explanation of the central paradox of the theory of confidence intervals and a suggestion with respect to its resolution in the context of the therapeutic trial. If we try to resolve the paradox on these terms we deduce a *prospective* calculus, and are led to an approach to statistical

* *e.g.* Sequential Analysis.

inference which drastically limits its practical scope and its mathematical interest. We are forced to admit that a primary problem in the comparative trial is the specification of the number of subjects which must be used, and to agree with the older school of statisticians that, for any results of conceivable value to accrue this number must be very large.

In the light of our earlier discussion this is not surprising. But it would be wrong to leave the impression that our attitude towards the place of statistics in therapeutics should be defined purely as a result of an elucidation of the logical credentials of its methodology. Were arbitrarily large samples available, the problem of statistical inference would become unimportant and we could reasonably proceed by identifying sample values with population values. But some of our earlier strictures on the utility of the statistical method would still remain operative. To this extent, therefore, it is these which constitute the more fundamental aspects of the case against statistical experimentation.

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COMPARATIVE EFFICACY OF HYPNOTICS

A SELF-CONTROLLED, SELF-RECORDED CLINICAL TRIAL IN NEUROTIC PATIENTS

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Although insomnia due to neurotic illness is one of the commonest of symptoms, reports on the comparative efficacy of hypnotics in man are rare. Werz and Homann (1939) and Cohen and Beecher (1951) made controlled comparative studies but were concerned with the sedative rather than the hypnotic effects of the drugs they used. Meyers, Cook, and Page (1940) investigated the hypnotic action of five drugs and a placebo in chronically ill patients, but did not attempt to compare the hypnotics with one another. A search through the literature of the past 20 years has not revealed any other relevant work. There are a number of probable reasons why the problem has not been studied more.

In the first place, there is wide individual variation in the response to a hypnotic drug. If it were also true that an individual's response to one hypnotic bore little relation to his response to another, then there would be no value in attempting to compare the efficacy of different hypnotics in a series of patients. Yet this point is one that does not seem to have been established, and on theoretical grounds we might expect that the *comparative* efficacy of different hypnotics would remain the same in different individuals provided the cause of the insomnia was the same.

In the second place, the conditions necessary for a controlled trial of hypnotics in man are not easily or commonly available, for such a trial requires a series of patients, all suffering from insomnia, under continuous night observation for at least several weeks, and, for preference, sufficiently co-operative and intelligent to give an account of how they slept. These conditions, however, may be fulfilled in hospital wards for the treatment of neurotic patients.

In the third place, so many efficacious hypnotics are available that to compare them might seem a work of supererogation. Indeed, in prescribing hypnotics the practitioner suffers an *embarras de richesse*, and in consequence either tends to confine himself to the use of one or two "favourite" hypnotics or administers a succession of newly-marketed drugs, the comparative efficacy of which has rarely been the subject of a controlled trial.

A satisfactory means of comparing the clinical efficacy of hypnotics would have two valuable uses. It would enable new hypnotics to be tested against long-established drugs, and it would permit a comparison of the efficacy of those already in common use. The common hypnotics are usually prescribed in a dosage which experience has shown as that which gives most patients reasonable relief from insomnia and at the same time is associated with a minimum of undesirable side-effects; it has not, however, been clearly established whether there is any significant difference in the efficacy of these drugs when given in their commonly prescribed dosage. If such differences could be established, then, other things being equal, it would be rational to administer the hypnotic which a controlled trial showed to be most effective.

Present Investigations

Hogben and Sim (1953) have outlined a method for determining the effect of treatment in low-grade morbidity by means of a self-controlled and self-recorded clinical trial. The present investigation was planned as an attempt to study, by this method, the comparative efficacy of hypnotics in patients suffering from neurotic insomnia.

In a self-recorded trial it is necessary to demonstrate the reliability of the self-record by checking it against an independent objective assessment. Two main checks were used in the present investigation, where the self-record was the patient's opinion of the quality of his night's sleep: the first, an external check, was the night-nurse's record of the patient's sleep; the second, an internal check, was achieved

by giving the patient the same hypnotic drugs in differently coloured capsules. If the patient's record is in close agreement with that of the night-nurse, and if in addition the recorded quality of sleep on a particular drug was the same whatever the colour of the capsules, then it would seem reasonable to accept the self-record as a reliable indication of the quality of the patient's sleep. The quality of sleep over a series of nights when the patient is receiving one drug could thus be compared with that when he is receiving another drug or a placebo, provided, of course, that the severity of insomnia remains the same during the different trials. There is no way of assuring this provision, but it might be assumed to hold if the different drugs are given in a random order and for a number of nights sufficient to smooth out the many chance factors that influence the quality of any particular night's sleep.

Material

Thirty females, all patients in a neurosis unit, were studied. A patient was admitted to the trial if she:

(*i*) complained of sleeping poorly or badly for the past few weeks or months (no attempt was made to distinguish between patients who had difficulty in getting off to sleep and those who woke early, for in the majority of neurotic patients no clear distinction of this nature is possible);

(*ii*) was able and willing to co-operate;

(*iii*) appeared to be a reliable witness (on this count, patients with low intelligence, with evidence of dementia, or with psychopathic or grossly hysterical personalities were excluded).

The thirty patients suffered for the most part from neurotic states in which anxiety and depression were the prominent symptoms. Their average age was 40, with a range from 18 to 73. They usually received 3 gr. amylobarbitone for the first two nights after admission and then were placed on the trial, remaining on it for as long as hypnotics were indicated or until they took their discharge. Patients who were on the trial for less than fourteen consecutive nights were excluded from the final assessment.

Administration of Hypnotics

Four separate comparative trials were made, as follows:

(*i*) *Trial A* (six subjects).—The effect of butobarbitone gr. 3 was compared with that of carbromal gr. 12 and also with that of a lactose placebo. The capsules containing these drugs were all identical in appearance, but half of the capsules were coloured red and half were coloured blue. They were administered in a pre-arranged random order (known only to the dispenser), such that over a period of eighteen nights a patient would receive each drug six times, three times in a red capsule and three times in a blue. The patients were not told they were receiving different types of drug; to minimize the chance of their noticing a difference in taste, the capsules were taken under the supervision of the night-nurse and the patients were asked to swallow them whole with a drink of water. To ensure that each patient had the correct capsules, these were sent from the dispensary in separate envelopes on which was written the name of the patient and the date on which the dose was to be taken.

(*ii*) *Trial B* (five subjects).—Sodium butobarbitone gr. 3 was compared with methylpentenol 0·3 g. and with a placebo. As methylpentenol is a liquid, it is administered in special soft elastic gelatine capsules. It was therefore necessary to administer the other drugs in a similar manner; to this end, arachis oil was used as a placebo, and the sodium butobarbitone was dissolved in propylene glycol (butobarbitone is insufficiently soluble for this purpose and therefore could not be used).

(*iii*) *Trial C* (thirteen subjects).—Sodium butobarbitone gr. 3 was compared with methylpentenol 0·5 g. No placebo control was used.

(*iv*) *Trial D* (six subjects).—Sodium butobarbitone gr. 3 was compared with carbromal gr. 12, without a placebo control. The drugs were administered in differently coloured capsules as in Trial A. As 3 gr. sodium butobarbitone is equivalent in barbiturate content to 2·72 gr. butobarbitone, we should expect to find that the efficacy of the barbiturate as compared with carbromal would be less in Trial D than in Trial A.

Record Charts

In general, a person will consider she has had a good night's sleep if she:

(*i*) got off to sleep quickly,

(*ii*) slept soundly for about 8 hours,

(*iii*) had no periods of wakefulness,

(*iv*) had no disturbing dreams,

(*v*) felt fresh on waking.

Any scheme that takes account of these factors should give an adequate record. The relative weight that ought to be attached to the factors is debatable, but probably not of great importance. The form of record chart used is shown overleaf. The mark scored by each possible answer is given in parenthesis. In an initial pilot study a question on dreaming was included, but subsequently omitted in the belief that the presence or absence of dreaming was probably not any indication of the efficacy of the hypnotic. It will be seen that a fully satisfactory night's sleep scores no marks, a very unsatisfactory one scores eleven. An adequate reason for halving the scores (toothache, for example) occurred only twice.

SLEEP RECORD

NAME..... DATE.....

1. Was your night's sleep—	*excellent*	(0)
	good	(1)
	fair	(2)
	poor	(4)
	bad	(6)
2. Did you get off to sleep—	*very quickly*	(0)
	normally	(0)
	with difficulty	(1)
3. Was your sleep—	*sound and uninterrupted*	(0)
	of average quality	(0)
	shallow and unsatisfying	(1)
	broken by long waking periods	(1)
4. On waking, did you feel—	*lively*	(0)
	normal	(1)
	stale	(2)
	"drugged" or confused	(3)

5. If you did not sleep well, was there any special reason? (If reason adequate, halve the score)

From the nurse's point of view, a patient slept well if she:

(*i*) was observed to be asleep for the greater part of the night,

(*ii*) was not restless.

The night-nurse kept a record which was scored as follows:

one mark for each half-hour awake between the hour of 10 p.m. and 7 a.m.;

one mark if there were one or two periods of night waking, two marks if more than two such periods;

one mark if a patient was recorded as "somewhat restless", two marks if "very restless".

If there was "considerable" (as opposed to "none" or "slight") disturbance in the patient's ward, one mark was subtracted. A satisfactory night's sleep would thus score no marks, a sleepless and restless night could score 20 marks.

If the records are reliable, then with one factor excepted the score from the nurse's record should show a high correlation with that from the patient's record. The excepted factor is the patient's estimate of how she felt on waking (it may be called the "hangover" score) as this has no counterpart in the nurse's record. Therefore, for the purpose of correlating the nurse's record against the patient's, the hangover score must be omitted.

Other Considerations

All the patients continued, during their period of trial, to have whatever methods of treatment seemed appropriate to their illness. In the course of treatment, most of the patients improved and many ceased to need hypnotics. It might be argued that the effects of a hypnotic cannot be properly evaluated while a patient is undergoing some relatively drastic form of treatment such as electroplexy (sixteen of the thirty patients had this during their hypnotics trial). However, as no patient goes without some form of treatment, and as the type of hypnotic was changed every night or every second night, it seemed justifiable to assume that the effects of the treatment on the insomnia would on the whole be evenly spread and would not seriously influence the comparative quality of the sleep obtained with the different drugs. This question is referred to again in the discussion.

Results

Table I (opposite) shows the patients' and nurse's average nightly scores in the four trials. Remembering that a low score means a good night's sleep, we see that in Trial A the scores obtained with butobarbitone are in every instance lower than those with the lactose placebo and, with two exceptions, lower than those with carbromal. Also, the carbromal scores are lower (in all but one instance) than the placebo scores. In Trial B, sodium butobarbitone scores are consistently lower than those with methylpentenol or the placebo, but there is little difference between the two latter. Consistency is again apparent in Trial C, where in every instance the sodium butobarbitone scores are less than those for methylpentenol, though the difference is less marked than in Trial B. In Trial D there is evidently little to choose between the two hypnotics.

Table II (overleaf) shows the scores in Trials A and D with the differently coloured capsules. It is evident that there is close agreement between the sets of scores obtained with red capsules and with blue capsules.

The "hangover" effects of the different drugs, as scored in the patient's self-record, are shown in Table III (overleaf). It appears that by this method of estimation none of the drugs in the dosages used caused any considerable "hangover" as compared with the placebos, though there is a slight tendency (in Trials A and D) for the scores on carbromal to be less than those on barbiturate.

It is evident from Table I that there is a wide variation between individual patients in the quality of sleep obtained. One way of considering the combined results in each trial is to allot equal weight to each patient's scores and this can be done by adjusting the barbiturate score to a fixed figure, say a hundred, for each patient and expressing the scores for other drugs as a percentage of this figure. When the scores obtained in this way are added together, the combined results for each trial are as shown in Table IV (overleaf). The comparative scores in each

TABLE I

AVERAGE NIGHTLY SCORES BY PATIENTS (SELF-RECORDED) AND NURSE WITH VARIOUS HYPNOTICS

	Trial	Subject	Number of Nights	Patient's Score			Nurse's Score		
				Butobarb.	Carbromal	Lactose	Butobarb.	Carbromal	Lactose
A	Butobarbitone gr. 3 Carbromal gr. 12 Lactose	1	19	1·60	2·29	3·57	1·80	2·50	3·00
		2	19	2·63	2·71	7·25	3·86	6·17	16·00
		3	22	1·40	2·00	2·71	3·57	4·88	6·00
		4	35	1·07	1·64	2·73	3·31	2·26	3·33
		5	35	2·00	1·92	3·80	5·40	6·00	6·40
		6	26	3·50	5·22	5·56	5·75	8·13	8·00
	Total	6	156	12·20	15·78	25·62	23·69	29·94	42·73
				Sod. butobarb.	Methyl-pentenol	Arachis Oil	Sod. butobarb.	Methyl-pentenol	Arachis Oil
B	Sod. butobarbitone gr. 3 Methylpentenol 0·3 g. Arachis Oil	1	14	1·50	2·25	—	4·17	5·75	—
		2	30	0·83	1·50	1·62	0·78	2·17	1·64
		3	18	0·33	0·86	2·00	2·83	3·00	4·60
		4	14	1·00	1·17	1·25	2·25	3·00	2·74
		5	26	1·13	5·27	4·57	6·00	8·18	7·75
	Total	5	102	4·79	11·06	(9·44)	16·03	22.10	(16·74)
				Sod. butobarb.	Methyl-pentenol	—	Sod. butobarb.	Methyl-pentenol	—
C	Sod. butobarbitone gr. 3 Methylpentenol 0·5 g.	1	13	1·83	2·29		4·00	5·15	
		2	14	1·14	2·79		1·57	4·43	
		3	26	1·25	1·50		3·58	4·07	
		4	14	1·00	2·00		2·80	4·29	
		5	33	1·18	1·63		3·89	4·81	
		6	15	1·17	1·50		3·83	5·14	
		7	19	1·20	2·25		2·25	3·08	
		8	30	3·24	4·31		3·60	4·00	
		9	33	2·82	4·75		3·40	4·31	
		10	33	2·71	3·63		5·63	6·63	
		11	36	2·06	2·42		3·00	3·80	
		12	41	3·12	3·25		8·31	9·20	
		13	18	3·16	3·53		6·08	7·62	
	Total	13	335	25·88	35·85	—	51·95	66·53	—
				Sod. butobarb.	Carbromal		Sod. butobarb.	Carbromal	—
D	Sod. butobarbitone gr. 3 Carbromal gr. 12	1	20	1·00	1·00		3·22	2·36	
		2	14	1·83	3·00		6·33	5·80	
		3	25	1·43	1·73		4·69	6·86	
		4	21	2·56	1·83		9·63	8·67	
		5	27	2·92	2·58		6·82	6·83	
		6	17	1·71	1·70		3·13	3·27	
	Total	6	124	11·45	11·84	—	33·82	33·79	—

trial are of the same order as those in Table I. Table IV enables us to make other deductions. From the results in Trial D, carbromal gr. 12 is about as efficacious as sodium butobarbitone gr. 3; but from Trial A, this dose of carbromal is rather less efficacious than butobarbitone gr. 3. Our confidence in the validity of the method is thus supported, for butobarbitone contains 10 per cent. more of the barbiturate radical than an equal dose of its sodium salt and ought, therefore, weight for weight, to be a rather more effective hypnotic. In the same manner, Trials B and C show what is to be expected: that 0·3 g. doses of methylpentenol are less effective than 0·5 g. doses, when each is compared against 3-gr. doses of sodium butobarbitone.

The degree to which the night-nurse's record checks that of the patient on any particular drug can be expressed as the correlation coefficient between the average nightly scores of the patients and of the nurse in each trial. This is shown in Table V. Further, the patients' average nightly scores on the nights they were receiving the barbiturate may be correlated, for each trial, with their scores when they received the other hypnotic; and the nurse's scores for the two drugs may be similarly correlated. These correlation coefficients are shown in Table VI The comparatively low correlation between the patients' scores for sodium butobarbitone and for methylpentenol in Trial B is entirely due to the high average nightly score of Subject 5 when on methylpentenol. With this exception, the correlations are of a high order, indicating that, although there was great

TABLE II

AVERAGE NIGHTLY SCORES BY PATIENTS AND NURSES WHEN THE SAME DRUGS WERE ADMINISTERED IN DIFFERENTLY COLOURED CAPSULES

The patients' "hangover" scores are also shown.

Trial	Subject	No. of Nights	Patients' Score		Nurses Score		Hangover Score	
			Red Capsules	Blue Capsules	Red Capsules	Blue Capsules	Red Capsules	Blue Capsules
A	1	19	2·56	2·34	2·28	2·39	2·00	2·00
	2	19	3·33	3·23	3·42	5·48	1·89	1·63
	3	22	1·33	2·23	3·46	5·81	1·48	1·40
	4	35	1·71	1·84	2·91	2·93	0·95	1·21
	5	35	2·61	2·18	5·57	5·94	1·92	1·89
	6	26	5·06	·440	7·86	7·11	2·92	3·00
	Total	156	16·60	16·22	25·50	29·66	11·16	11·13
D	1	20	1·00	1·00	2·50	3·07	0·33	0·19
	2	14	2·25	2·00	5·83	6·02	2·50	3·00
	3	25	1·00	1·95	4·92	5·70	1·00	1·27
	4	21	2·25	2·17	9·34	9·58	1·00	1·00
	5	27	2·60	3·04	6·43	7·07	2·50	2·33
	6	17	2·00	1·70	3·63	3·03	1·00	1·25
	Total	124	11·10	11·86	32·65	34·77	8·33	9·04

individual variation in the response to a given hypnotic, the *comparative* activity of two hypnotics was practically the same in the different subjects.

DISCUSSION

Two main criticisms may be made of this investigation: that the number of subjects in each trial is small and that over half the subjects were receiving electroplexy during their period of trial. The numbers are admittedly small; but even in a neurosis ward suitable subjects are not very easily found, for in addition to the requirements mentioned above the patient must be prepared to have a few nights of poor sleep (when receiving the placebo) and his sleep must not be consistently excellent on the drugs tested or there would be no difference to record. However, even with the small number of subjects, the results are sufficiently consistent to make the investigation seem worth reporting. The second criticism is more serious, as electroplexy may disturb a patient's judgment. In Trial A, however, only one of the six subjects (Subject 3) received electroplexy, yet all the trials show a similar degree of consistency. On the other hand, the fact that ten of the thirteen subjects in Trial C and four of the six in Trial D received electroplexy might possibly explain the rather lower correlation between the scores of the patients and of the nurses in these two trials.

If these criticisms are met, then the results of the trial allow of four main conclusions:

(1) Reasons for believing the method to be reliable may be summarized as follows:

(*i*) the patients' self-record of the quality of their sleep showed a high positive correlation with the night-nurses' record;

(*ii*) the patients' scores when they received a series of drugs in red capsules were practically the same as when they received the same drugs in blue capsules;

(*iii*) neither patients nor nurses knew when a hypnotic or a placebo was administered; but the quality of sleep recorded on placebo nights was consistently the poorer;

(*iv*) compared against a fixed dose of carbromal, the mean quality of sleep of a series of patients receiving butobarbitone was somewhat better than that of patients receiving the same weight (and therefore a smaller barbiturate equivalent) of sodium butobarbitone; and a similar relation held between patients receiving 0·5 g. and 0·3 g. of methylpentenol when tested separately against a fixed dose of sodium butobarbitone.

TABLE III

AVERAGE NIGHTLY "HANGOVER" SCORE OF PATIENTS ON DIFFERENT HYPNOTICS

Trial	Subject	No. of Nights	Hangover Score		
			Butobarb gr. 3	Carbromal gr. 12	Lactose
A	1	19	2·00	2·00	2·00
	2	19	2·13	1·14	2·25
	3	22	1·86	1·13	1·29
	4	35	0·92	1·18	1·08
	5	35	2·00	2·00	2·00
	6	26	3·00	2·89	3·00
	Total	156	11·91	10·34	11·62
			Sod. butobarb. gr. 3	Methyl-pentenol 0·3 g.	Arachis Oil
B	1	14	1·33	1·38	—
	2	30	1·00	1·55	1·92
	3	18	1·00	1·00	1·20
	4	14	1·00	1·00	1·00
	5	26	2·13	2·09	2·00
	Total	102	6·46	7·02	(6·12)
			Sod. butobarb. gr. 3	Methyl-pentenol 0·5 g.	—
C	1	13	2·67	3·00	
	2	14	2·00	1·86	
	3	26	1·83	1·61	
	4	14	1·00	1·13	
	5	33	1·00	1·13	
	6	15	1·17	1·25	
	7	19	1·07	1·29	
	8	30	1·47	1·54	
	9	33	1·53	2·19	
	10	33	2·22	2·38	
	11	36	1·41	1·58	
	12	41	1·17	1·10	
	13	18	1·69	1·93	
	Total	335	20·23	21·99	—
			Sod. butobarb. gr. 3	Carbromal gr. 12	—
D	1	20	0·00	0·45	
	2	14	3·00	0·45	
	3	25	1·14	1·17	
	4	21	1·00	1·00	
	5	27	2·38	2·22	
	6	17	1·29	1·20	
	Total	124	8·81	8·64	—

TABLE IV

MEAN FOR EACH TRIAL OF THE AVERAGE NIGHTLY SCORES RECORDED BY THE PATIENTS AND BY THE NURSE

In calculating the means, each subject's record has been given equal weight by expressing her score on each drug as a percentage of her barbiturate score

Trial	No. of Subjects	Drug	Patients	Nurse
A	6	Butobarbitone gr. 3 ..	100	100
		Carbromal gr. 12 ..	132	126
		Lactose	212	185
B	5	Sod. butobarb. gr. 3 ..	100	100
		Methylpentenol 0·3 g.	237	159
		Arachis oil	325	155
C	13	Sod. butobarb. gr. 3 ..	100	100
		Methylpentenol 0·5 g.	148	139
D	6	Sod. butobarb. gr. 3 ..	100	100
		Carbromal gr. 12 ..	107	101

TABLE V

CORRELATION BETWEEN AVERAGE NIGHTLY SCORES RECORDED BY THE PATIENTS FOR EACH DRUG AND THE EQUIVALENT SCORE RECORDED BY THE NIGHT-NURSE

Trial	Correlation Coefficient
A	+ 0·85
B	+ 0·82
C	+ 0·51
D	+ 0·63
For all trials combined	+ 0·70

TABLE VI

CORRELATIONS BETWEEN THE PATIENTS' AVERAGE NIGHTLY SCORES WHEN RECEIVING THE BARBITURATE AND WHEN RECEIVING THE ALTERNATIVE HYPNOTIC; AND BETWEEN THE EQUIVALENT SCORES RECORDED BY THE NIGHT-NURSE

Trial	Correlation Coefficient	
	Patients' Scores	Nurses' Scores
A	+ 0·91	+ 0·86
B	+ 0·47	+ 0·97
C	+ 0·85	+ 0·94
D	+ 0·59	+ 0·89

For these reasons it would appear that the self-controlled, self-recorded clinical trial could satisfactorily be used for testing a new hypnotic and comparing its efficacy in combating neurotic insomnia with that of a given dose of an established hypnotic.

(2) Although there was wide individual variation in response to any particular hypnotic, the comparative response to two hypnotics remained almost constant among the subjects in the trials. This constancy of comparative response to hypnotics does not appear to have been clearly established hitherto. If it is a fact, then in general we can no longer suppose that some patients respond well to one particular hypnotic while others may respond better to a different hypnotic.

(3) In the doses given, the hypnotic drugs administered caused no appreciable subjective feeling of "hangover". Goodnow and others (1951) and Felsinger, Lasagna, and Beecher (1953) have nevertheless shown that there may be impairment of physiological performance after barbiturate in the absence of subjective sensations. The patients' estimates of how they felt on waking was in some respects the least satisfactory part of the trial. One or two of the patients with long-standing neurotic disability seemed to feel they owed it to themselves always to complain of waking up "drugged" whenever they believed they had been given a hypnotic. This is an attitude of mind which might explain the statement of Goodman and Gilman (1955) that "hangover from relatively small hypnotic doses (of barbiturates) occurs especially among neurotic patients".

(4) The results suggest that carbromal in a dose of 12 gr. is less efficacious than butobarbitone gr. 3 but has approximately the same efficacy as sodium butobarbitone gr. 3. Carbromal in a dose of 10–15 gr. would thus appear to be a hypnotic of considerable potency and has perhaps been unduly neglected; the National Formulary (1955), for example, states that it is "a weak hypnotic", while Goodman and Gilman (1955) dismiss the monoureides as "somewhat disappointing due to their feeble depressant effects".

Methylpentenol, on the other hand, in a dose of 0·3 g. proved little better than a placebo, and 0·5 g. was sitll considerably less effective than 3 gr. sodium butobarbitone. Hirsh and Orsinger (1952), who administered methylpentenol to 276 patients in doses up to 0·5 g., concluded that it was most effective in "simple insomnia" and less so in agitated states; a similar conclusion was reached by Chevalley and others (1952), and the present trial tends to support these findings.

SUMMARY

(1) This paper describes a self-controlled and self-recorded clinical trial of the comparative efficacy of three common hypnotic drugs in thirty patients suffering from neurotic insomnia.

(2) The reliability of the self-records was checked by several independent methods.

(3) This type of trial appears to yield reliable results and could be used to test the efficacy of new hypnotics.

(4) Butobarbitone gr. 3 was shown to give more relief from insomnia than carbromal gr. 12, which in turn was more efficacious than methylpentenol 0·5 g.

My grateful thanks are due to Mr. E. F. Wellington, dispenser at Barrow Hospital, for his kindness in preparing and dispensing the trial capsules; and to the night-nurses in the neurosis ward for their conscientious co-operation.

Addendum

After this paper had been written, the author's attention was drawn to the work of Straus, Eisenberg, and Gennis (1955). By a self-controlled and self-recorded trial, they have compared the hypnotic activity of an antihistamine with that of phenobarbitone and a placebo. Their method of recording the quality of sleep was somewhat different, and their statistical analysis was more complex than that used in the present investigation; but the underlying principle and general conclusions as to the suitability of the method for comparing the action of hypnotics were the same.

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SOCIAL MEDICINE STUDIES BASED ON CIVILIAN MEDICAL BOARD RECORDS

IV. OCCUPATIONAL AND PHYSICAL CHARACTERISTICS OF MEN WITH CERTAIN DIGESTIVE DISORDERS

BY

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In a large sample of wartime Civilian Medical Board (C.M.B. *passim*) records it was found that 1·2 per cent. of the men examined were unfit for military service because of a peptic ulcer, and a fur- 0·25 per cent. because of other digestive disorders (Stewart, Webb, and Hewitt, 1955a). In addition to this loss of manpower at the screening stage there was a further heavy loss from the same causes among men accepted for military service: peptic ulcer ranked second only to psychiatric disorder as a reason for discharge from the army (War Office, 1948). Thus digestive disorders caused more serious disability than any of the other disease groups so far dealt with in this series (foot defects, varicosities, and skin diseases). In some ways this means that peptic ulcer is less suitable for study through massed medical records. The condition may, for example, make a patient lose weight or change his job, so that it becomes difficult, in a statistical analysis, to distinguish between cause and effect. Despite this difficulty it was felt that an analysis of the peptic ulcer records both by occupation and by physique could be of value, provided the results were interpreted with caution. To this study has been added a similar analysis of other types of dyspepsia and of appendicitis.

Material and Methods

The material and methods are the same as those described previously (Stewart and others, 1955b), and the Standardized Prevalence Ratios (S.P.Rs *passim*) make full allowance both for regional and age differences in recorded prevalence.

At the outset of the investigation it was arbitrarily decided to code as duodenal ulcers all those not specified as "gastric". It is estimated that, as a result, rather less than one-eighth of the so-called duodenal group may, in fact, have been gastric. In a small number of cases a man was recorded as having both a gastric and a duodenal ulcer, and such cases were counted in *both* the disease groups. The label "Other Dyspepsia" covers all men recorded as complaining of indigestion in whom no specific cause was found; it excludes cases diagnosed as cholecystitis, colitis, or liver disease.

A history of appendicitis was coded only when an appendicectomy scar had been noted at the examination.

Prevalence

Age and Area.—The numbers of records of the four digestive disorders obtained from the three areas, together with crude prevalence rates per 1,000 are shown in Table I.

Table I

RECORDED PREVALENCE OF FOUR DIGESTIVE DISORDERS AMONG MEN EXAMINED AT THREE CIVILIAN MEDICAL BOARD CENTRES (1941–44)

Type of Disorder		Essex	Leicester	Northampton	Total
Gastric Ulcer	Number	45	157	53	255
	Rate per 1,000	3·8	6·8	3·4	5·0
Duodenal Ulcer	Number	120	257	130	507
	Rate per 1,000	10·0	11·1	8·2	10·0
Other Dyspepsia	Number	1,001	963	512	2,476
	Rate per 1,000	83·7	41·5	32·5	48·6
Appendicitis	Number	349	901	442	1,692
	Rate per 1,000	29·2	38·8	28·0	33·2

The recorded prevalence of peptic ulcer, even after allowing for age differences, was about 30 per cent. lower than that found by Doll, Jones, and Buckatzsch (1951). To some extent the discrepancy may be accounted for by geographical factors, since the male death rate from peptic ulcer (particularly gastric ulcer) is known to be well above average in Greater London, where most of the material used by Doll and others (1951) was collected (Registrar-General, 1951). The discrepancy may also be related to occupational differences; for example, the prevalence of ulcer in the relevant age-groups of the earlier enquiry would have been

* In receipt of a grant from the Medical Research Council.

some 5 per cent. lower if, like the C.M.B. sample, it had not included any doctors. But the possibility must also be borne in mind that a considerable number of men with peptic ulcers may not have been included in the wartime "census of fitness for military service".* Although the prevalence rate of ulcers was lower than that previously recorded (Doll and others, 1951), the number of cases available for analysis was more than twice as great.

After standardizing for age there were no perceptible differences between the three regions in the recorded prevalence of duodenal ulcer, and no difference between Essex and Northampton with respect to gastric ulcer or appendicitis. Leicester had relatively high rates for both these conditions, which may indicate regional differences in morbidity. On the other hand, the apparent excess of dyspepsia cases in Essex is likely to be spurious, since this (less serious and less clearly defined) disease group clearly gave greater scope for personal differences between doctors, and Essex was known to have kept more comprehensive records than the other two areas.

All areas showed the expected age-distributions, namely, a continuous steep rise from the early twenties in the case of peptic ulcers, and other dyspepsia, and an approach to a "ceiling" prevalence at about the age of forty for appendicitis.

SOCIAL CLASS.—The main features of the social distribution of the four digestive disorders are shown in Table II.

For peptic ulcer as a whole there was a regular, though not statistically significant, gradient from a low level in Social Classes I and II to a high level in Social Class V. This gradient clearly had its origin in the gastric ulcers, which were more than twice as common in Social Class V as in Social Classes I and II. (No S.P.R. is shown in Table II because of the small numbers involved: eighteen cases recorded against 27·88 "standard" cases.) Since this excess was found in the social class for whom diagnostic opportunities are, reputedly, inferior, it seems likely to be genuine. By contrast with gastric ulcer, duodenal ulcer showed no social variation. This difference is similar to the result obtained by Doll and others (1951), whose site classification was more precise, and it tallies with the mortality figures of the last 35 years (Registrar-General, 1954). It therefore seemed justifiable, in the following occupational analysis, to calculate S.P.R.s for the gastric and duodenal groups separately.

TABLE II
STANDARDIZED PREVALENCE RATIOS FOR FOUR DIGESTIVE DISORDERS IN THE REGISTRAR-GENERAL'S SOCIAL CLASSES

Social Class	Peptic Ulcer	Gastric Ulcer	Duodenal Ulcer	Other Dyspepsia	Appendicitis
I II	} 93	D†	107 {	115 91	129 123**
III	97	96	97	101	98
IV	109	99	115	101	106
V	115	153***	96	100	85*
χ^2	3·627	11·975	1·845	3·020	16·239
Degrees of Freedom	3	3	3	4	4
$P(\chi^2)$	$>$·30	$<$·01	$>$·50	$>$·50	$<$·01

† Less than twenty cases of gastric ulcer recorded in these classes.

In this and Tables III and IV, individual ratios are considered to differ significantly from 100 if the associated contribution to the sum of χ^2 would have been significantly large in a test based on 1 d.f. They are marked by asterisks as follows:

* $P < ·05$; ** $P < ·01$; *** $P < ·01$; **** $P < ·001$

No social variation could be detected in the prevalence of other dyspepsia, but appendicitis showed a definite gradient with the highest prevalence in Social Class I. Each of the three areas reproduced this excess in the upper classes. This finding is of particular interest since, as Table III shows, the variation seemed to be restricted to men over 30 years old in 1941 (*i.e.* men born before the first world war). It is just possible that there is a genuine age difference in the social distribution of appendicitis, like that previously found in the case of acne (Stewart and others, 1955c). An alternative explanation is that the distribution of cases among the older men was determined at a time when appendicitis was mainly an "upper class" diagnosis, and that it was not till later that the operation was more widely performed among the working classes. A similar change can be traced in American figures for tonsillectomy (Downes, 1954). But even if it is reasonable to suppose that the frequency of appendicectomy and tonsillectomy have been influenced by fashion, this can hardly explain recent changes in appendicitis

TABLE III
STANDARDIZED PREVALENCE RATIOS FOR APPENDICITIS BY SOCIAL CLASS

Social Class	Age Groups (yrs)	
	17–29	30–44
I and II	107	127****
III	97	98
IV and V	108	91
χ^2 for 2 d.f.	1·239	13·885
$P(\chi^2)$	$>$·50	$<$·01

* Permanently disabled men (other than those certified as blind, mentally defective, insane, or permanently crippled by the loss of a limb) were subject to the National Service Registration Acts, but in practice some of them were not examined by C.M.B.s. It was, for instance, possible for a doctor's certificate of *permanent* disablement to be accepted in lieu of an examination and for no record to be kept of the case.

mortality. Thus in 1921 and 1931 appendicitis, *as a cause of death*, was more than twice as common in Social Class I as in Social Class V. By 1950 the difference had almost disappeared (Registrar-General, 1954). Taken together, the mortality and moribidity figures suggest that we are, in fact, dealing with an authentic example of the gradual spread through the whole population of a disease formerly restricted to the higher income groups.

OCCUPATION.—The principal findings of the occupational analysis are shown in Table IV.

TABLE IV

STANDARDIZED PREVALENCE RATIOS FOR FOUR DIGESTIVE DISORDERS, BY OCCUPATION

Occupational Group	Peptic Ulcer	Gastric Ulcer†	Duodenal Ulcer	Other Dyspepsia	Appendicitis
Professional and Administrative	99	D	113	90	118
Commercial ..	101	101	101	94	108
Clerical	102	D	104	93	109
Boot and Shoe ..	83	94	78*	90	89
Textile	97	103	94	91	96
Metal Manufacture	116	D	131	107	104
Trade not defined‡	97	D	97	116*	111
Warehouse ..	105	D	126	99	113
Transport ..	87	D	86	110	96
Building and Agriculture ..	75*	D	69*	98	81**
Unskilled ..	114	159***	92	106	81*
Other	128***	111	136***	105	115*
χ^2 for 11 d.f. ..	18·768	10·782	23·389	16·043	26·231
$P(\chi^2)$	<·01	>·30	<·02	<·20	<.·01

‡ Occupation as recorded common to several skilled trades (e.g. fitters, cutters, inspectors, etc.)

† The seven Occupational Groups marked D each yielded less than twenty records of gastric ulcer, the number recorded being in each case significantly less than the "standard" number.

For peptic ulcer as a whole the deviations from standard prevalence were of questionable significance, and those for gastric ulcer merely reflected the previous analysis by social class.

The results for duodenal ulcer, however, showed a definite association between occupation and prevalence. There were deficiencies of cases among builders, and among agricultural workers, which together yielded the lowest S.P.R. (69) shown in the table. "Boot and shoe" workers also had a significant deficiency of duodenal ulcers, which was found to originate in Northampton (occupational S.P.R. for Northampton alone, 66) rather than in Leicester (S.P.R. 96). But the largest departures from the standard prevalence of duodenal ulcer were in the heterogeneous group of "Other Occupations" which had a combined S.P.R. of 136. Subdivision of this group showed that the excess was concentrated in two small sub-groups, each of which had, a significantly high rate for both types of peptic ulcer:

(*i*) *Painters and Decorators* (Registrar-General's Main Order XV) had fifteen recorded cases of duodenal ulcer compared with a standard number of 6·85 (χ^2 with Yates' correction, 8·543; $P \simeq 0{\cdot}003$); 25 years ago, when "Painters and Decorators" had more deaths from occupational lead poisoning than any other group, they also had a high death rate from peptic ulcer (Registrar-General, 1938), but this was due to deaths from gastric rather than duodenal ulcer.

(*ii*) *Entertainments and Sport* (Registrar-General's Main Order XXI) yielded six cases of duodenal ulcer compared with a standard number of 1·81 ($P \simeq 0{\cdot}006$*).

A third subdivision of the "Other Occupations" group in Table IV with an excess of duodenal ulcers was "Personal Service" (S.P.R. 139), but this could not be regarded as statistically significant.

The occupational analysis for "Other Dyspepsia", like the social class analysis, failed to show any significant variation, but there were some definite occupational differences in the prevalence of appendicitis. To some extent these may have been due to the social class gradient already discussed, but this can hardly explain the low S.P.R. of 81 for "Building and Agriculture". The "Agricultural" workers, whom one might have expected to have a low rate for appendicitis, in fact showed their full number of standard cases, and it was the "Builders" (S.P.R. of only 68) who brought down the rate for the combined group. As in the case of duodenal ulcer the most interesting departures from standard prevalence of appendicitis were in the smaller occupations making up the "Other Occupations" group in Table IV. Two definitely high rates were for "Personal Service" (S.P.R. 165, $P \simeq {\cdot}01$), and for "Entertainments and Sport" (eight cases, compared with a standard number of 2·41, $P \simeq {\cdot}002$). The rate for "Painters and Decorators" was again suggestively high.

PHYSIQUE AND PEPTIC ULCER.—S.P.R.s for gastric and duodenal ulcer, taken separately and together in relation to height, weight, and body build, were calculated by the method previously described (Stewart and others, 1955b). All the results are plotted together in the Figure (overleaf), the measurements for height, weight, and body build being expressed in standard deviations above and below the mean. Because of missing measurements the number of cases was about 100 less than in the previous section. The greatest range of variation was found among the weight groups. Thus peptic ulcers were

* When the standard number of cases was very small the probability was estimated by summing terms of the appropriate Poisson distribution and making the usual correction for continuity.

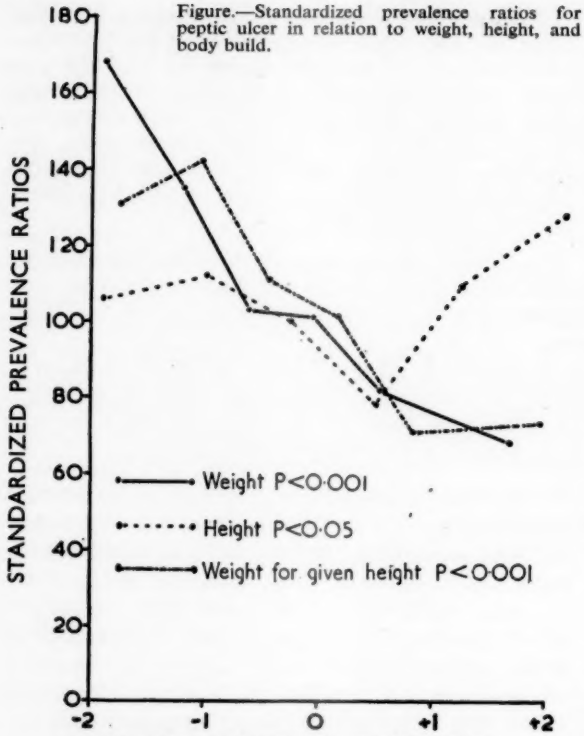

Figure.—Standardized prevalence ratios for peptic ulcer in relation to weight, height, and body build.

twice as common among men weighing less than 110 lb. as among those who weighed 150 lb. or more. However, this wide contrast in prevalence corresponds to a very small difference between the *average* weight of the ulcer subjects and that of a group of controls matched for region and age (4·35 lb. for duodenal ulcers alone, 4·78 lb. for gastric ulcers). A difference of this magnitude might easily be a residual effect of the illness rather than an indication that ulcer subjects are, initially, below average in weight. Similar considerations apply to the relatively high prevalence of ulcer among men who, as judged by the index Ht $\div \sqrt[3]{}$Wt, were light for their height. It was of interest here to find a strong similarity between the distribution of the two ulcer groups. This is illustrated by Table V in which the S.P.R.s for each extreme of the Ht $\div \sqrt[3]{}$ Wt scale are compared (these extremes correspond to the highest and lowest pairs of points shown in the Figure).

TABLE V

Build	Gastric Ulcer	Duodenal Ulcer
Slender Extreme ..	149·8	134·0
Stocky Extreme ..	77·5	69·2
Ratio $\frac{\text{Slender}}{\text{Stocky}}$ ..	1·93	1·94

One might have been tempted to infer from such a degree of similarity that the gastric and duodenal groups had not been adequately separated in the original coding, were it not for the fact that the two diseases had already been shown to differ in their social distribution. If, as seems likely, the tendency to be underweight is merely the result of having an ulcer, there would of course be no reason to expect a difference between the two groups.

The observed variation along the height scale, though it did not reach the same high level of statistical significance as that for weight and body build, is difficult to explain. There appears to be a minimum prevalence towards the middle of the scale, both short men and tall men being more liable than those just above average height. This finding is principally due to the height distribution of the men with *duodenal* ulcers and would have been overlooked if the analysis had been conducted solely in terms of average measurements.

DISCUSSION

Clinical teaching has long favoured a picture of the peptic ulcer patient as the paradigm of asthenic physique, in sharp contrast with the gallbladder patient who is taken to represent the pyknic, or stocky type of build. But evidence for the first of these views is neither as extensive nor as unanimous as is generally believed. The authorities on constitutional medicine most widely quoted are Kretschmer (1944), Draper (1930), Sheldon, Stevens, and Tucker (1940), and Sheldon, Hartl, and McDermott (1949), whose studies in the field of peptic ulceration seem to have three features in common:

(*i*) Their observations on ulcer patients are not adequately controlled by parallel observations on typical, healthy persons;

(*ii*) They do not consider that any peculiarity in the appearance or measurements of the ulcer subjects could be a *result* of illness;

(*iii*) Their clinical series do not run to much over 100 cases (compared with more than 600 in the present study).

Kretschmer (1944) using a method of physique classification that is in essence subjective, reported that 71 per cent. of 59 stomach ulcer patients were of the "leptosome" (slender) type, compared with 38 per cent. of 1,000 other surgical patients. On the other hand, in one of Draper's earlier studies it is admitted that, although the gastric ulcer male differs widely from representatives of other disease groups, he shows only a "very slight divergence from the form of the general population" (Draper, 1930). In a later work gastric and duodenal ulcer patients are

said to possess very similar physical characteristics, and, although slightly above the average of a hospital series in stature, they are said to be 3·75–4·75 kg. (8¾–10½ lb.) lighter in body weight (Draper, Dupertuis, and Caughey, 1944). The sample of duodenal ulcer patients studied by Sheldon and others (1949) was found to contain a preponderance of endomorphic—mesomorphs (*i.e.*, persons whose principal physical characteristic was muscularity), but so were the other disease groups studied at the Presbyterian Hospital, New York, and it is left an open question whether the duodenal subjects really differed in physique from the population from which this hospital drew its patients (Sheldon, Hartl, and McDermott, 1949). As against Draper and his group, Sheldon and his colleagues are confident that gastric ulcer patients can be distinguished from duodenal ulcer patients by an absence of gynandromorphy, a finding which is "so consistent that it is almost possible to predict the location of the lesion from a careful examination of the physique" (Sheldon, Stevens, and Tucker, 1940).

In the present study it has not been possible to attempt the detailed characterization of physique practised by these authors. Our figures confirm the clinical impression that men who have already developed a peptic ulcer tend to be thinner than other men, and show that they have an unusual height distribution which cannot be interpreted as an effect either of their illness or of treatment. But, taken together with the other evidence, our results suggest that the relevance of physical type to the ulcer diathesis has been exaggerated. The aetiological significance of "ulcer personality" has also been called into question by Kellock (1951) who failed to find any difference between the childhood histories of 250 duodenal ulcer patients and a control group of patients with other diagnoses.

Summary

An analysis has been made of the social class and occupational distribution of men, who, at the time of their pre-service medical examination, had a peptic ulcer, complained of dyspepsia, or exhibited an appendicectomy scar. Amongst the previous findings confirmed by this study were an excess of gastric ulcer in the poorer classes and a comparatively even social class prevalence of duodenal ulcers. A clear excess of peptic ulcers was noted among "Painters and Decorators" and among workers in "Entertainment and Sport." A relatively low prevalence of duodenal ulcer was found among "Boot and Shoe" factory workers and among workers in "Building and Agriculture."

For men born before the first world war, but not for younger men, there was a clear association between appendicectomy and membership of the well-to-do classes. Builders had a low rate for appendicitis, but there was a definite excess of patients with this disease among workers in "Entertainment and Sport" and in "Personal Service".

The prevalence of peptic ulcer was also examined in relation to height, weight and the body build index Ht $\div$ $\sqrt[3]{\text{Wt}}$. Although men who had developed an ulcer were found to be somewhat underweight, the results do not prove that any particular type of body build is associated with liability to peptic ulcer. On the other hand the unusual height distribution of duodenal ulcer subjects seems to require explanation. Some previous work in this field is discussed.

We should like to record our thanks to the Nuffield Provincial Hospitals Trust and the Medical Research Council who contributed generously toward the expenses of the investigation.

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BODY MEASUREMENTS OF PRE-SCHOOL CHILDREN

BY

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The only serial study of body measurements of young children in the British Isles known to the author is that of Low (1952) on Aberdeen children born in 1923–25. The present paper is intended to fill this gap. A knowledge of body measurements is necessary for the proper design of equipment and furniture; it is also necessary for the study of physical types and for the interpretation of weight records.

A set of measurements was taken from age 1 to age 5 years on a small group of children forming part of a national survey of weights and heights under the guidance of a committee including representatives of the Ministry of Health, Ministry of Education, British Paediatric Association, Society of Medical Officers of Health, and research bodies in association with the public health authorities of eleven areas.

These measurements extend the data on body measurements already published for children of school age, 5 to 18 years (Hammond, 1953). Weight and length at birth are also given. Standard deviations of the measurements are included for comparison with Low's figures in order to assess the significance of the differences (whether regional or secular). Since the measurements were taken on the same children each year, the mean growth represents the difference between the means for each year. The standard deviations of the annual growth increases are also given in order to show within what limits a year's growth in any measurement may be expected to lie for any particular child or group.

The measurements are intended as a guide to the relations between weight or height and other body measurements, so that if these two measurements are known for any group, by making use of the relations between the mean body measurements given in this paper and that of Low, it should be possible to estimate fairly closely the expected group dimensions in other measurements.

Sample

The sample consisted of all the babies in the above national survey, born at Leeds between September and December, 1949. The first year's measuring was undertaken on 451 children (214 boys and 237 girls). The follow-up for the next 4 years was only possible on those children born in December; this reduced the numbers to approximately seventy children measured each year. (From 2 years onwards the measurements were taken 2 months after the child's birthday, but the results have been adjusted (by linear interpolation) to correspond to the birth date). In any voluntary follow-up study of growth the subjects continuing to co-operate after a number of years tend to represent a superior group. In order to minimize any bias due to this factor, we visited the homes of those children whose mothers were unable or unwilling to bring them to a central clinic for measurements. Even so, weather conditions and illness caused the numbers each year to vary (as indicated in Table I, opposite). All measurements were retained for the cross-sectional means, but the increment data are based only on actual gains shown by individuals on successive measuring occasions. As a result of visiting the homes the group which was followed-up was found to differ little in its initial measurements compared with the whole group and the measurements can be accepted as representative of the whole group.

Measurements and Measuring Techniques

All babies were weighed and measured without clothing, the weighing machines used at the different clinics for the yearly measurements being checked by the local authority. Birth weights were obtained by a special inquiry from the records of hospitals or midwives, the mother's statement alone not being accepted. The fourteen measurements additional to weight were chosen as the result of a preliminary trial which the author undertook with two specially selected health visitors (Miss B. England and Miss O. Prentis). After a week's training, they then measured the larger sample at the age of one year. During the measuring I spent 3 days visiting the homes with the two health visitors to check the measurements. As babies are easily upset by measuring it was not possible to check the results rigorously by duplicating the measurements at this stage, but I was satisfied that the techniques had not altered between the training period and my visit. I myself undertook all the measuring at the follow-up examinations during the next 4 years. Details of the measurement techniques are given in the Appendix. Various checks were made on the measuring, including duplicate measurements, and the results are presented below.

Accuracy of Measurements.—An indication of the accuracy of the measuring techniques during the earlier years is given by the agreement between the measurements taken and the weights and heights which were available from the local authority's routine measuring, and also from the internal consistency of the measurements. (For example, sitting height and leg length measurements when added together should show a constant relation to total length measurement.)

TABLE I

MEAN MEASUREMENTS OBTAINED AND THEIR STANDARD DEVIATIONS BY SEX

Age (yrs)			Birth		1		2		3		4		5	
Numbers		Boys	214		214		44		24		34		30	
		Girls	237		237		45		35		40		37	
Measurements			Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
Weight (kg.)		Boys	3·38*	0·58	10·35*	0·94	12·39*	1·15	14·10*	1·21	16·11*	1·90	17·83*	2·07
		Girls	3·34*	0·51	9·94*	1·19	11·97*	1·42	14·21*	1·47	16·31*	1·73	18·12*	2·13
Height (cm.)		Boys	48·5	2·01	73·3	3·07	84·3	3·18	93·6	4·14	101·0*	3·75	107·7*	4·02
		Girls	47·8	2·12	72·0	2·57	83·7	3·23	94·0	3·42	101·6*	4·35	108·1*	4·72
Sitting Height (cm.)		Boys	—	—	45·4**	2·06	50·5	2·03	54·5	1·79	58·0	2·36	60·9*	2·19
		Girls	—	—	44·6**	1·86	49·9**	2·03	54·7	2·51	57·9	2·14	60·6	2·29
Leg Length (cm.)		Boys	—	—	29·5	1·67	35·6	1·65	40·5	1·98	44·5	2·15	48·4	2·45
		Girls	—	—	29·4	1·64	35·6	1·63	40·8	1·97	45·5	2·45	49·4	2·95
Breadth (cm.)	Chest	Boys	—	—	13·7	0·95	14·8	0·93	16·0	1·08	16·4	0·84	17·3	1·03
		Girls	—	—	13·5	0·91	14·6	0·81	15·6	0·85	16·1	0·88	17·0	0·89
	Inter-iliac	Boys	—	—	11·6	0·87	—	—	13·4	0·82	14·3	1·10	15·4	0·92
		Girls	—	—	11·4	0·82	—	—	13·3	0·83	14·3	0·70	15·3	1·05
	Inter-trochanteric	Boys	—	—	—	—	16·2	0·72	(17·2)	—	18·6*	0·98	19·2*	0·72
		Girls	—	—	—	—	16·6*	0·74	(17·3)	—	18·7*	0·83	19·2*	1·06
	Inter-acromial	Boys	—	—	17·2*	1·29	19·4	0·98	20·7	1·33	22·2	1·14	23·6	1·18
		Girls	—	—	16·9*	1·25	19·2	0·96	20·9	1·11	22·0	1·32	23·5	1·24
	Knee	Boys	—	—	5·51	0·44	6·46	0·33	6·89	0·37	7·25	7·38	7·47	0·31
		Girls	—	—	5·36	0·40	6·34	0·36	6·66	0·39	7·06	0·40	7·37	0·45
	Ankle	Boys	—	—	3·47	0·42	4·30	0·24	4·61	0·34	4·95	0·37	5·13	0·29
		Girls	—	—	3·36	0·37	4·18	0·26	4·48	0·22	4·82	0·24	5·09	0·26
Girth (cm.)	Upper-arm	Boys	—	—	15·8	1·23	16·5	0·92	16·3	1·07	16·6	0·91	16·6	0·95
		Girls	—	—	15·5	1·19	16·1	1·01	16·3	1·08	16·7	1·31	16·9	1·32
	Chest	Boys	—	—	—	—	—	—	52·3	2·11	54·3	2·18	55·1	2·30
		Girls	—	—	—	—	—	—	51·2	2·43	53·1	2·39	54·4	2·56

* mean significantly greater than Aberdeen group. ** significantly smaller (2 s.d.)

The following comparative results were obtained:

Measurements	Age (yrs)	Mean (constant) Difference (cm.)		S.d. Difference (cm.)	
		Boys	Girls	Boys	Girls
(1) Recumbent Height minus Standing Height	1	0·93	0·94	0·99	1·14
(2) Local Authority Survey minus Standing Height	2	0·64	0·31	2·11	1·56
(3) Difference Leg Length plus Sitting Height minus Standing Height	1	1·60	2·01	1·69	1·79
	2	1·77	1·78	1·50	2·00

At 1 year the recumbent length is greater than the standing height on average by about 1 cm., and the standard deviation of the differences is also about 1 cm. If there had been any considerable error in either measurement, the variability of the differences would have been greater than this. This standard deviation of differences actually includes variation due to errors of measurement and also variation in the true differences between recumbent and standing heights. As these two influences can be presumed to be independent, the total variance is the sum of their contributions and the error variance will therefore be less than this. Similarly the variance of the difference between the sum of constituent measurements and total length includes true differences in the relation as well as error variance, and hence the above figures represent the extreme level of possible error. As a reasonable approximation we can say that the measured heights, stem lengths, and leg lengths, can be presumed to be within 1 cm. of the true measurements in roughly two-thirds of the cases or within 2 cm. in 95 per cent. of the cases. At the 5th year when nine sets of duplicate measurements were taken, the errors were found to be less than this, the results being as follows:

Measurements		Standard Deviation of Duplicate Measurements (cm.)	Percentage Total Variance
Length	Arm Length	0·2	1
	Standing Height	0·60	2
	Sitting Height	0·7	10
	Leg Length	1·3	25
Girth	Arm	0·1	1
	Knee	0·1	1
	Chest	1·3	25
Breadth	Inter-iliac	0·1	1
	Ankle	0·05	2
	Inter-trochanteric	0·2	6
	Hand	0·1	10
	Knee	0·1	10

TABLE II
MEAN GROWTH INCREASES AND STANDARD DEVIATIONS BY SEX

Age (yrs)			0–1		1–2		2–3		3–4		4–5	
			Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.	Mean	S.D.
Weight (kg.)		Boys	6·97	1·02	2·04	0·59	1·71	0·50*	2·01	0·82	1·72	0·82
		Girls	6·60		2·03		2·24		2·10		1·81	
Height (cm.)		Boys	24·8	2·47	11·0	1·70	9·3†	1·28*	7·4†	1·25*	6·7	1·28*
		Girls	24·2		11·7†		10·3†		7·6†		6·5	
Sitting Height (cm.)		Boys	—	—	5·1	1·62	4·3	1·14	3·5†	2·17	2·9	1·30
		Girls	—		5·3		4·8†		3·2†		2–7	
Leg Length (cm.)		Boys	—	—	6·1	1·56	4·9	1·65	4·0	1·40	3·9	1·18
		Girls	—		6·2		5·2		4·7		4·9	
Breadth (cm.)	Chest	Boys	—	—	1·1	1·03	1·2	0·87	0·4	0·65	0·9	0·82
		Girls	—		1·1		1·0		0·5		0·9	
	Inter-iliac	Boys	—	—	—	—	—	—	0·9	0·78	1·1	0·67
		Girls	—		—		—		1·0		1·0	
	Inter-trochanteric	Boys	—	—	—	—	(1·0)	—	(1·4)	—	0·6	0·57
		Girls	—		—		(0·7)		(1·4)		0·5	
	Inter-acromial	Boys	—	—	2·2	1·12	1·3	0·78*	1·5	0·91	1·4	0·70
		Girls	—		2·3†		1·7		1·1		1·5	
	Knee	Boys	—	—	0·95	0·38	0·43	0·28	0·36	0·26	0·22	0·20
		Girls	—	—	0·98		0·32		0·40		0·31	
	Ankle	Boys	—	—	0·83	0·33	0·31	0·22	0·34	0·18	0·18	0·21
		Girls	—		0·82		0·37		0·34		0·27	
Girth (cm.)	Upper-arm	Boys	—	—	0·7	0·93	−0·2	0·57	0·3	0·71	0·0	0·52
		Girls	—		0·6		0·2		0·4		0·2	
	Chest	Boys	—	—	—	—	—	—	1·9	1·77	0·8	1·54
		Girls	—		—		—		1·9		1·3	

† significantly greater than Low's figure for Aberdeen children (2 s.e.).
* standard deviation of growth significantly *lower* than for Aberdeen children (2 s.e.).

These measuring errors could affect some individual annual increments appreciably, but the mean measurement increases would be unlikely to be affected since the errors were random and, therefore, tended to cancel each other. The error data are scarcely extensive enough to warrant making any adjustment to the observed variance of the annual growth increases to allow for the error variance.

RESULTS

INCREASE IN SIZE WITH AGE.—The mean measurements and their standard deviations at each year are shown in Table I. The mean growth increases with their standard deviations are shown in Table II.

Comparison between the Present Group and Low's Group of Aberdeen Children (*Low*, 1952).—The present group is bigger than Low's group in practically all measurements from birth onwards, though only the results asterisked are statistically significant. At age 4 the average child in the Leeds group is almost the equivalent of the 5-year-old child in Low's group. This difference may be partly regional, but it is more probably accounted for by the time interval separating the two groups. Thus Weir (1952) has shown that Glasgow 5-year-old children, are, on average, 1 kg. heavier and 4 cm. taller than children of the same area 20–25 years ago. The only measurement in which the Leeds children are significantly smaller than Low's group at any age is in sitting height at the 1st and 2nd year. This could be due to a difference in the techniques of measuring.

The growth patterns shown in Table II are interesting since they show a continuous deceleration in the growth of the lengths and ankle and knee breadths from birth to 5 years, whereas weight growth and growth in trunk breadths tend to remain more constant after the large rise from birth during the first and second years. The first year's growth in weight and height exceeds the total growth from 2–5 years.

Comparison with Council School Children aged 5 years.—In order to make a detailed comparison with the council school children (Group C) of age 5+ studied earlier (Hammond, 1953), additional measurements included in the school children's survey were also taken on the Leeds group at age 5*. The comparison is shown in Table III (opposite).

* The Leeds figures are adjusted to age 5 years 6 months to make a direct comparison possible.

TABLE III
LEEDS CHILDREN COMPARED WITH COUNCIL SCHOOL CHILDREN BY SEX
(Central age 5 years 6 months)

Measurement		Boys		Girls	
		Leeds Group	Council School "C" Group	Leeds Group	Council School "C" Group
Weight (kg.)		18·69	18·45	19·02	18·41
Height (cm.)	Standing	110·0	108·0	110·3*	107·8
	Sitting	62·3	61·4	62·1*	60·5
Length (cm.)	Leg	54·5*	52·3	54·9*	52·7
	Arm	46·1*	45·1	45·8*	44·2
	Foot	17·80*	17·21	17·78*	16·81
Breadth (cm.)	Foot	6·66	6·79	6·64	6·86
	Hand	5·79	5·82	5·67	5·72
	Inter-iliac	15·9*	14·9	15·9	14·7
	Inter-trochanteric	19·5	19·3	19·5	19·5
	Inter-acromial	24·3*	23·3	24·2*	23·2
Girth (cm.)	Arm	16·6*	15·9	17·0	16·0
	Knee	21·2	21·0	21·9*	20·9
	Chest	55·5	56·2	55·0	54·3
	Waist	52·0	52·5	51·9	51·7
	Hip (seat)	58·5	—	60·2	58·5

* Difference significant at 5 per cent. level.

The school children were drawn mainly from poor areas of industrial towns in the North of England.

These results show that the Leeds children were somewhat larger than council school children from poor areas in nearly all respects and significantly so in many of the length and breadth measurements. This was to be expected, since the Leeds group was drawn from good as well as poor areas and contained children from all social groups, whereas the council school group was drawn only from the poorest areas of industrial towns and consisted almost entirely of Social Classes III to V.

Variability of Measurements.—The standard deviations of attained measurements are very similar for Low's group and the present group.

The standard deviations of annual growth,* calculated from Low's data, are given in Table IV for comparison with the present results (shown in Table II).

TABLE IV
STANDARD DEVIATIONS OF ANNUAL GROWTH FROM BIRTH TO 5 YEARS CALCULATED FROM LOW (1952)

Age (yrs)	0–1	1–2	2–3	3–4	4–5
Weight (kg.)	0·97	0·75	0·83	0·77	0·64
Height (cm.)	2·68	2·20	2·05	2·17	1·92
Sitting Height (cm.)	1·78	1·22	1·39	1·25	1·28
Inter-trochanteric Breadth (cm.)	0·89	0·80	0·62	0·64	0·62
Inter-acromial Breadth (cm.)	1·48	1·02	1·10	0·83	0·64
Chest Girth (cm.)	2·32	1·59	1·55	—	—

Despite the slightly greater average growth in the Leeds group, the variability is on the whole somewhat less than for Low's group, especially in height.*

From the growth figures relative to their standard deviations it can be seen that many children will show no measurable increase during the year in some dimensions. Arm girth, for example, shows negligible increase from 2–5 yrs (largely because of the decrease in subcutaneous tissue) and 5–10 per cent. of children can be expected to show no increase over a year in acromial breadth, iliac breadth, knee and ankle breadths, whilst in chest breadth and girth up to 25 per cent. of children will be expected to show no annual increase during certain years.

RELATIONS BETWEEN MEAN MEASUREMENTS (WITH COMPARISONS BETWEEN PRE-SCHOOL CHILDREN AND SCHOOL CHILDREN)

Although the mean measurements differ according to age and for different groups, as, for example, for the Leeds group and Low's Aberdeen children, there may be constant relations between measurements when the general size difference is allowed for. A simple device for showing this is to plot measurements against each other, *e.g.* weight against height, in order to see whether the Leeds and Aberdeen children of the same height have the same weight, the same inter-acromial measurement and so on.

Weight according to Height.—When the weights and heights of the pre-school children are plotted on the same graph as that of the school children which showed a straight-line relation between the logarithm of weight and height† (Fig. 1, Boys, overleaf), the points for the means for Years 3, 4, and 5 fall closely upon this line. For Years 1 and 2 the weights are relatively somewhat greater for height, and below 1 year there is a quite different relation between weight and height. In the case of Low's group the points tend to fall below the line, since these children are relatively slight for their height (or alternatively they are about 1–1·5 cm. taller for corresponding weights).‡ The girls (not shown) follow the same tendency as the boys, having the same weight for height relation as the school children at ages 3–5 and being somewhat heavier for the corresponding height at ages 1 and 2.

* The standard deviations given are the averages of the standard deviations obtained for each sex, in order to even out fluctuations and enable a better comparison to be made between groups.

* Low's variability for growth in height appears to be unduly augmented by measuring errors in a few cases, thus five children have the highest growth increase in the group during one year followed by the lowest growth in the next year (or the converse); further, the figures for stature are not confirmed by the supra-sternal height results. In the present group two such extreme growth results are found and these also are not confirmed by growth in related measures (leg length and sitting height). Any doubtful values would increase the apparent variability of growth without materially affecting the mean.

† Eqn. Log. Weight (kg.) = 0·0082 Height (cm.) + 0·38.

‡ This difference, especially at years 1, 2, and 3, is probably attributable to a difference in measuring technique since length was measured recumbent, and this was found to be greater than the standing height measurement (p. 4).

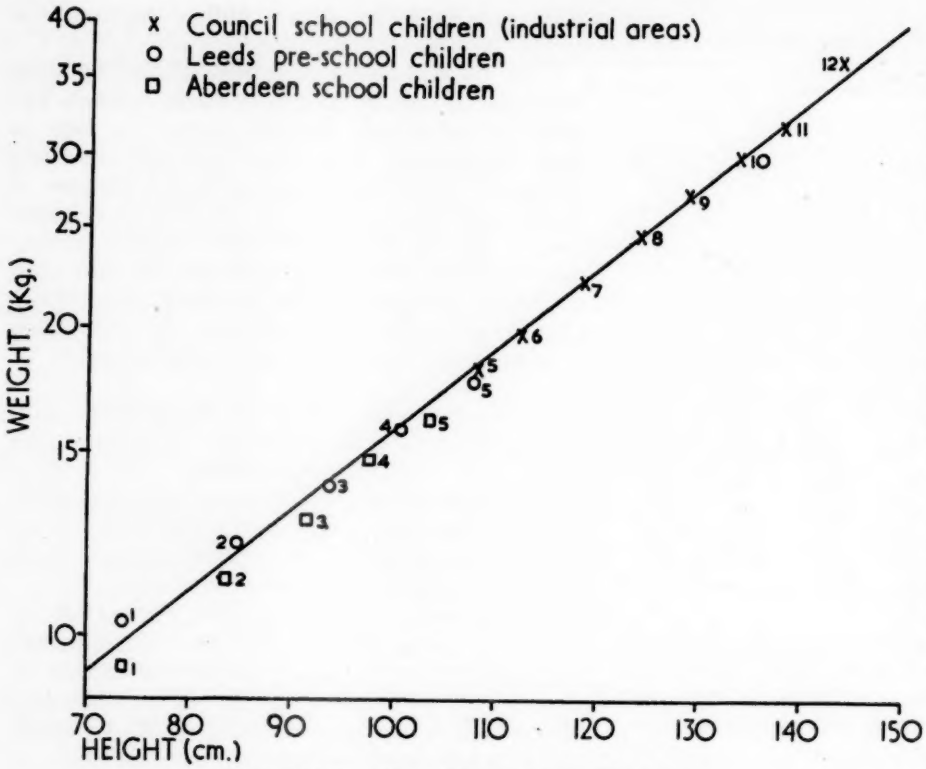

FIG. 1.—Relation of log weight to height at different ages.

Chest Girth according to Weight.—Chest girth was only measured at ages 3, 4, and 5 in the Leeds pre-school children, but over this range the relation to weight follows that of the council school children,

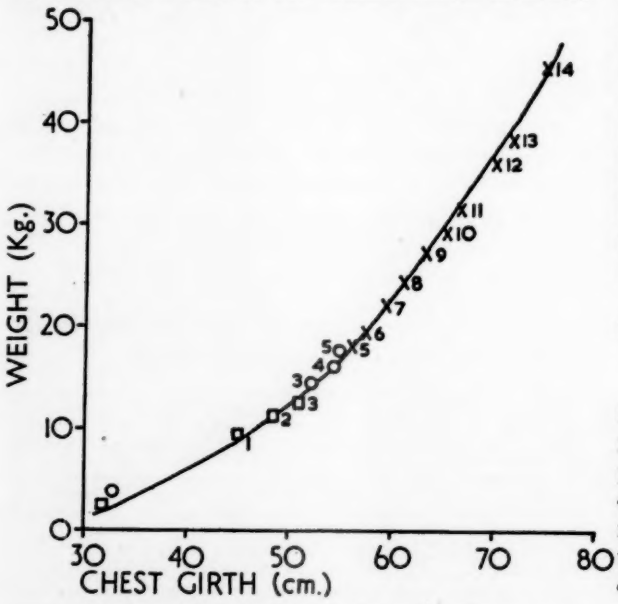

FIG. 2.—Relation of weight to chest girth at different ages.

i.e. the same curve can be projected backwards from the school children's range or forwards from the pre-school group (Fig. 2). Low's group also follows the same curve.

Inter-acromial and Inter-trochanteric Breadths according to Height.—The relations between height and inter-acromial or inter-trochanteric breadths are also in the form of a straight line and parabolic curve respectively, and these again coincide with the relations shown by the council school children (Fig. 3, Boys, opposite).

The girls also conform to the school children's pattern. Low's group follows a parallel straight line giving a slightly greater breadth measurement (0·5 cm.) for a given height.

The constancy of these relations, besides confirming the previous work on school children, suggests that there can be fairly wide differences in absolute measurements according to the nature of the sample studied, but that, if basic criteria of weight and height are known, other mean measurements can be predicted from these with reasonable accuracy by reading off the values from the graphs. It should be noted that this constancy of relations between means does not imply that the means show the same *proportions* at different ages, but only that the pattern of change is a predictable one.

Prediction of an Individual's Measurements from Height and Weight

The actual values of chest girth, etc., as predicted from height and weight are given by the appropriate regression equations. The closeness with which the predictions are likely to apply in individual cases depends on the correlations between the measurements concerned and also on their standard deviations. The actual distribution of weights for different heights will be presented when the analyses of the more extensive data of the National Survey,

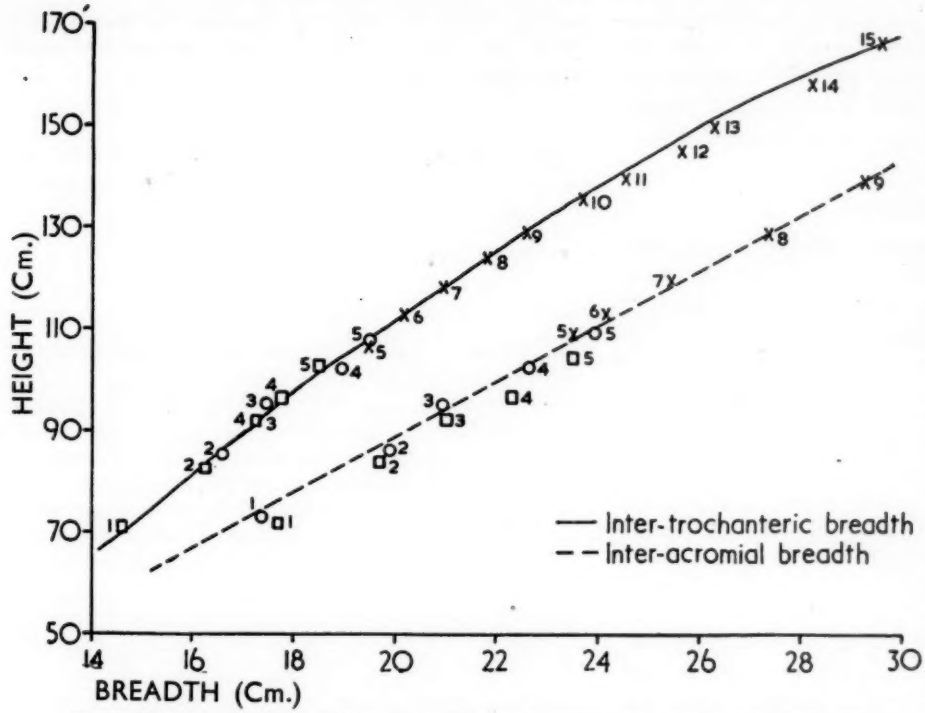

FIG. 3.—Relation of height to inter-trochanteric and inter-acromial breadths at different ages.

from which the present group was drawn, are completed.

The mean within-year correlations between heights and lengths or breadths, or between weights and girths, for various groups of council school and independent school children as well as for the present sample are given in Table V. The numbers in each yearly group range from 100 to 120.

and height. Thus, nearly 80 per cent. of the variance in lengths and 20–50 per cent. of the variance in breadths can be predicted from height, whilst weight covers some 60–70 per cent. of the variance in girths (the reduction is actually in the proportion $1-r^2$ times the full variance, where r is the correlation between predicted and predictor measurements). To take an example, a child of 5 years 6 months would be expected to have a hip girth of $58{\cdot}5 \pm 4{\cdot}6$ cm. with a probability of 95 per cent. However, if his weight is known, the variability is reduced to $\pm 2{\cdot}5$ cm. If the child is known to be one standard deviation above average in weight, his most probable deviation in hip girth will be 0·82 s.d. or 1·9 cm. ($r \times$ s.d.). The value of 60.4 cm. for hip girth for a child weighing 20·8 kg. (+ 1 s.d.) agrees almost exactly with the point on the mean weight-hip girth curve (not shown) corresponding to this weight. The graphs of mean measurements in relation to each other can be used

TABLE V

WITHIN-YEAR CORRELATIONS BETWEEN HEIGHT AND WEIGHT AND OTHER BODY MEASUREMENTS

Correlation of Height with		Sitting Height	Leg Length	Foot Length	Inter-iliac Breadth	Inter-trochanteric Breadth	Inter-acromial Breadth	Ankle Breadth	Knee Breadth	Chest Breadth
Age (yrs)	0–5	·770	·721	·888	·400	·427	·452	·568	·374	·359
	5–10	·869	·927	·806	·542	·727	·634			
	5–13	·882	·897	·776	·522	·697	·616	·689	·659	·496
	11–13	·848	·917	·830	·540	·711	·557			

Correlation of Weight with		Chest Girth	Waist Girth	Hip Girth	Arm Girth
Age (yrs)	0–5	·706	·650	·822	·715
	5–10	·804	·717	·797	·762
	5–13	·867	·802	·854	·791
	11–13	·824	·783	·814	·847

Both the pattern of correlations and the relations between the means are very constant for the different ages studied, and therefore we can safely use the values to give a general indication of the usefulness of predictions of different measurements from weight for individuals as well as for groups, though with less precision, especially if the child differs markedly from the mean for his age.

The present study is a longitudinal one essentially concerned with growth, and therefore the numbers are rather small to justify setting up regression equations for different combinations of measurements for predicting an individual's unknown from his known measurements. However, the fact that the results of the present study agree closely with those from Low's group and the school children's

groups in respect of the relations obtaining between the measurements (as shown in Figs 1–3 and the correlations above) indicates that the data can be used with greater confidence for graphical predictions than the small numbers might suggest.

Summary

The present paper reports the successive body measurements of a group of Leeds children from the age of 1 to 5 years. The sample included all social grades and the measurements are intended to extend the data on school children aged 5–18 years (Low, 1952).

The mean measurements at each year are compared with Low's figures for a group of Aberdeen children and also with the 5-year old group of the author's survey of school children (Hammond, 1953). The Leeds children are bigger than Low's group by almost one year's growth at ages 4 and 5 years, and they are also bigger than the author's group of school children.

The curves of weight in relation to height and chest girth, and of height in relation to inter-acromial and inter-trochanteric breadth derived from the school children's survey apply to this pre-school group when extended backwards. From these curves, mean body measurements can be predicted if weight and height are known. Aberdeen children showed slight differences from these curves.

Since the study was a longitudinal one, the differences in the measurements at each age represent true average growth increases. The standard deviations of the growth increments at each age are given.

Sets of correlations between height and breadths or lengths and between weight and girths at different ages show that the relations between body measurements at different ages are remarkably constant; they indicate the closeness within which any individual's measurements can be predicted from his height and weight.

I wish to acknowledge the generous help given by Dr. Catherine M. Gray and the Public Health Department for Leeds in making all the arrangements for the clinics and home visits, and also for allowing the two health visitors, Miss B. England and Miss O. Prentis, to undertake the first year's measuring. I am also most grateful to the last-named for the conscientious way in which they carried out the measuring in the home and laid the foundations of the goodwill which enabled the study to be continued with the minimum of defections. Mr. G. Taylor, Chief Education Officer, kindly allowed us to continue the study during the children's first year at school. Finally, the co-operation of the mothers (many of whom were working) who brought their children each year to clinics or made arrangements for them to be cared for whilst being visited, should not pass unmentioned.

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APPENDIX

Techniques

(1) *Weight.*—Nude weight, nearest ounce recorded.

*(2) *Standing Height.*—One person measures, using a Martin anthropometer, whilst the other supports the baby, holding the ankles so that the heels remain on the ground and pressing back slightly to prevent the knees from bending, and at the same time holding the head upright. Checked by comparison with total length obtained routinely at one year in the clinics, using a head board and movable foot board (recumbent).

(3) *Stem Length.*—Length from top of head to ground, when the baby was sitting with legs horizontal and back supported vertically.

(4) *Leg Length.*—From top of trochanter to external malleolus, using steel tape.

(5) *Chest Breadth.*—At nipple height, distance between lateral vertical lines through middle of arm pits, using wooden sliding calipers with curved arms.

(6) *Inter-iliac Breadth.*—Width between anterior superior spines of iliac bones using pelvimeter.

(7) *Inter-trochanteric Breadth.*—Using sliding calipers.

(8) *Inter-acromial Breadth.*—Palpating outer edges of acromia and using pelvimeter.

(9) *Knee Breadth.*—At head of tibia, using an industrial sliding guage fitted with broad plates on the jaws—firm contact.

(10) *Ankle Breadth.*—Intercondylar breadth using calipers—firm contact.

(11) *Upper-arm Girth.*—Maximum girth using $\frac{1}{4}''$ tape measure with very light pressure so as not to produce indentation, arm raised slightly from side of body.

(12) *Chest Girth.*—At nipple height, using $\frac{1}{4}''$ tape measure with minimum tension required to prevent tape slipping, measurement taken as near as possible midway between inspiration and expiration.

Additional measurements were taken at age 5 for comparison with the results from the school children presented in the previous communication (Hammond, 1953).

* In the trial, total length recumbent was found to be no more accurate than standing height at age one year, and as it constrained the baby much longer and was the one measurement to which the babies objected most strongly it was excluded in favour of standing height.

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A NOTE ON THE OBSTETRIC EFFECTS OF WORK DURING PREGNANCY

BY

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For women having their first babies, gainful employment—at least during the first half of pregnancy—is nowadays common in all levels of society in Great Britain (Martin, 1954). The largest series of first births to be studied in respect of work during pregnancy was taken from the sample of all babies born in England, Wales and Scotland between the third and ninth of March, 1946 (Royal College of Obstetricians and Gynaecologists, 1948). These cases have since been the subject of a detailed report (Douglas, 1950) in which it is shown that premature babies were unusually frequent among two groups of primigravidae: those who went out to work during the later months of pregnancy, and those who lacked domestic help. In neither group was the excessive prematurity rate attributable to maternal age, economic status, or illegitimacy. Two Scottish surveys (Ferguson and Logan, 1953; Illsley, Billewicz, and Thomson, 1953) have also examined the association between prematurity rates and work during pregnancy. In both these inquiries the prematurity rate was higher among women who followed gainful employment than among housewives, but in one (Illsley and others, 1953) the authors concluded that the actual length of time worked had not affected birth weight. A more recent study (Martin, 1954) suggests that employment during pregnancy is not associated with risk of premature delivery. No stillbirths were included in this survey.

The present study, which attempts to answer the question whether pregnant women should be encouraged to follow gainful employment, is based on 1,318 single first births notified to the administrative county of Northamptonshire during 1952. In respect of employment during pregnancy these women fall into three groups:

(1) Housewives not gainfully employed (780)
(2) Women gainfully employed for less than 28 weeks (285)
(3) Women gainfully employed for more than 28 weeks (253)

There were 55 perinatal deaths* and 89 babies who weighed less than 5½ lb. at birth.

* Stillbirths and neonatal deaths.

In respect of both perinatal mortality and incidence of prematurity working mothers appeared to be at a significant disadvantage as compared with housewives, and women who continued working for long periods appeared to be worse off than those who retired relatively early (Table I).

TABLE I
OBSTETRIC HISTORY OF HOUSEWIVES AND WOMEN GAINFULLY EMPLOYED

Mortality, Prematurity, etc.	Housewives (per cent)	Women gainfully employed: less than 28 wks (per cent)	Women gainfully employed: 28 wks or more (per cent)	All Cases (per cent)	Significance
Perinatal Death Rate	2·8	4·9	7·5	4·2	$P<·01$
Incidence of Prematurity (*i*) Birthweight 5½ lb. or less	4·7	8·4	11·1	6·8	$P<·01$
(*ii*) Delivered 15 or more days before expected date	9·9	12·3	14·6	11·3	$P<·20$
Incidence of Toxaemia of Pregnancy	14·1	14·4	15·4	14·4	—
Frequency of Instrumental Delivery	12·9	10·5	9·1	11·7	—
Incidence of Congenital Defect	0·4	0·7	1·6	0·7	—
Number of Cases	780	285	253	1,318	

The three groups of mothers were also compared in the following respects:

Maternal Age.—470 women were over 25 years of age when the baby was born; in this group there were proportionately more deaths and more premature babies than in the group of younger women. The group with the highest percentage of women over 25 years (42·4 per cent.) was the housewives; corresponding proportions among women employed less than or more than 25 weeks were 26·7 and 24·9 per cent. respectively. This excludes maternal age as the reason why working mothers had higher infant mortality and prematurity rates.

Maternal Physique.—Recent surveys have shown that short stature and obesity in the mother are

associated with an increased risk of foetal and neonatal death (Baird and Illsley, 1952; Stewart, Webb, and Hewitt, 1955). In both these respects there was virtually no difference between the three working groups.

Toxaemia of Pregnancy.—The sample included 190 women who showed definite evidence of toxaemia of pregnancy. These cases were so evenly distributed between the three groups (Table I) that it is difficult to believe that employment influenced this particular hazard of pregnancy. In fact, eight of the deaths had been ascribed by the certifying doctors to toxaemia, and of these three were in the younger and five (all housewives) in the older age group.

Instrumental Delivery.—The proportion of instrumental deliveries was slightly (but not significantly) smaller in the two working groups than in the other group (Table I). The eleven deaths actually ascribed to injury or difficult delivery included five among housewives and three in each of the working groups.

Congenital Defects.—Only nine deaths were due to congenital defects; the incidence is higher for housewives than for the other two groups but the difference is not significant (Table I).

Social Class.—The perinatal death rate and the incidence of prematurity were more than three times as high among wives of semi-skilled and unskilled manual workers (Classes IV and V) as among wives of professional men and business executives (Classes I and II) (Table II). There was also a higher proportion of housewives (not gainfully employed) in the latter group. Within groups defined by social class there were differences in the obstetrical results of employed women and housewives. Only in Social Class III (the largest of the groups analysed) were these differences statistically significant, and then only in respect of the incidence of prematurity.

TABLE II
SOCIAL CLASS OF HOUSEWIVES AND WOMEN GAINFULLY EMPLOYED

Mortality, Prematurity	Social Class	Housewives (Per cent.)	Women Gainfully Employed: less than 28 wks (per cent.)	Women Gainfully Employed: 28 wks or more (per cent.)	All Cases (per cent.)	Significance
Perinatal Death Rate	I and II	1·9	—	—	1·5	—
	III	2·6	5·3	6·1	4·0	—
	IV and V	3·9	6·0	12·3	6·1	—
Incidence of Prematurity (birthweight 5½ lb. or less)	I and II	2·6	3·3	5·9	3·0	
	III	4·0	8·5	10·4	6·5	$P<·02$
	IV and V	7·8	10·4	13·7	9·6	
Number of Cases	I and II	156	30	17	203	—
	III	421	188	163	772	—
	IV and V	203	67	73	343	—

Civil Status at Time of Conception.—In the sample there were 48 illegitimate births and 162 legitimate births conceived two or more months before marriage. Since it had been shown that the risk of perinatal death was virtually the same for these two classes (Stewart and others, 1955), they were combined and compared with all other births in the sample (see Table III). The perinatal death rate and incidence of prematurity were significantly higher for the premarital than for the postmarital conceptions. There was also a higher proportion of housewives in the latter group. Within groups defined by civil status at the time of conception there were differences in the obstetrical results of employed women and housewives. In the largest group (postmarital conceptions) the differences in incidence of prematurity reached a 5 per cent. level of significance.

TABLE III
CIVIL STATUS AT TIME OF CONCEPTION

Mortality, Prematurity	Conception	Housewives (per cent.)	Women Gainfully Employed: less than 28 wks (per cent.)	Women Gainfully Employed: 28 wks or more (per cent.)	All Cases (per cent.)	Significance
Perinatal Death Rate	Premarital	4·8	9·7	12·9	9·5	—
	Postmarital	2·6	3·6	4·8	3·2	—
Incidence of Prematurity (birth weight 5½ lb. or less)	Premarital	6·3	14·5	14·1	11·9	
	Postmarital	4·6	6·7	9·5	5·8	$P<·05$
Number of Cases	Premarital	63	62	85	210	—
	Postmarital	717	223	168	1,108	—

Matched Samples.—In a group of 143 women employed for less than 28 weeks and an equal number of housewives, matched for age, social class, and civil status at time of conception, the incidence of prematurity was 4·9 per cent. for the employed women and 2·1 for the housewives. In an equivalent group of 152 women employed for more than 28 weeks and 152 housewives, the corresponding rates were 7·3 and 1·3 per cent.

Stated Causes of Death.—In addition to 28 deaths ascribed to toxaemia, injury and congenital defects, there were nine cases in which a definite cause of death was given by the certifying doctors, and

eighteen in which vague terms (such as prematurity and atelectasis) were used. The deaths included 25 babies who weighed 5½ lb. or less at birth, distributed as follows:

Nine housewives (1·2 per cent.);
Five women employed less than 25 weeks (1·8 per cent.);
Eleven women employed more than 25 weeks (4·3 per cent.).

Taken in conjunction with the other findings, this result suggests that the reason why women employed during pregnancy had less successful pregnancies than housewives was that they gave birth to more small babies whose survival rate was low. Occupations of mothers with dead babies did not appear to be either exceptionally dangerous or unusually exacting even when compared with housework.

SUMMARY

An examination of 1,318 records of first pregnancies suggests that attempts to combine housework and paid work increase the risk of prematurity and consequently of stillbirth and neonatal death.

I am indebted to Dr. C. M. Smith, Medical Officer of Health for the County of Northamptonshire, and to his staff of midwives and health visitors who have done most of the field work connected with this survey.

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CHANGING PATTERN OF MORTALITY IN ENGLAND AND WALES

III. MORTALITY IN OLD AGE

(a) Picture Disclosed by Annual Specific Rates

BY

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Previous contributions to this series have dealt with infant mortality and with maternal mortality (Taylor, 1954; Taylor and Dauncey, 1954).

The aim of this survey is to examine what ages are of special interest *vis à vis* the conservation of life, and to complete it we have still to deal with old age. An appropriate definition of what we mean by the term old age in this context will emerge later. It will clear the ground for subsequent discussion if we first ask how many people reach extreme age.

The current Life Table presented in Table I is based on data given in the Registrar General's one per cent. sample of the 1951 census. It sums up present day mortality for a fictitious population of 100,000 males and 100,000 females born in 1952, each subject to the average mortality rate of 1950–52. Of these approximately 90,000 males and 93,000 females would reach age 45, 52,000 males and 68,000 females would reach age 70, and 8,000 males and 18,000 females would reach age 85. These estimates are based on death rates at the present time. We shall later attempt to show the most probable effects of future changes in mortality. In the 20 years since 1931 the population over 80 years of age in England and Wales has approximately doubled—from 208,000 to 406,000 for females and from 114,000 to 221,000 for males; but very little of this change is due to reduced mortality at high ages. Nearly all the expansion is attributable to the particular age structure of the community,

TABLE I
LIFE TABLES

Table ..	English No. 10 (1930–32)				Abridged England and Wales, 1950–52			
Sex ..	Male		Female		Male		Female	
Age (yrs)	$1x$ Survivors	$\overset{o}{e}_x$	$1x$ Survivors	$\overset{o}{e}_x$	$1x$ Survivors	$\overset{o}{e}_x$	$1x$ Survivors	$\overset{o}{e}_x$
0	100,000	58·74	100,000	62·88	100,000	66·33	100,000	71·47
1	92,814	62·25	94,545	65·48	96,725	67·57	97,478	72·31
2	91,394	62·21	93,273	65·37	96,488	66·74	97,266	71·47
3	90,794	61·62	92,711	64·76	96,353	65·83	97,153	70·55
4	90,394	60·89	92,334	64·03	96,255	64·90	97,072	69·61
5	90,069	60·11	92,024	63·24	96,180	63·95	97,011	68·65
10	89,023	55·79	91,082	58·87	95,799	59·19	96,782	63·81
15	88,360	51·19	90,420	54·28	95,541	54·34	96,603	58·92
20	87,245	46·81	89,383	49·88	95,089	49·59	96,291	54·11
25	85,824	42·54	88,133	45·55	94,436	44·92	95,854	49·34
30	84,416	38·21	86,792	41·22	93,745	40·32	95,296	44·62
35	82,885	33·87	85,353	36·87	92,934	35·56	94,624	39·91
40	80,935	29·62	83,690	32·55	91,885	30·94	93,764	35·26
45	78,357	25·51	81,660	28·30	90,319	26·43	92,544	30·69
50	74,794	21·60	78,958	24·18	87,540	22·19	90,636	26·28
55	70,041	17·89	75,290	20·23	82,960	18·27	87,852	22·04
60	63,620	14·43	70,204	16·50	75,852	14·75	83,670	18·01
65	54,899	11·30	63,046	13·07	65,599	11·67	77,350	14·28
70	43,361	8·62	53,144	10·02	52,324	8·99	67,792	10·94
75	29,665	6·43	40,040	7·45	36,718	6·75	54,190	8·06
80	16,199	4·74	24,869	5·46	21,042	4·92	35,953	5·88
85	6,377	3·50	11,594	4·00	8,062	3·83	17,731	4·35
90	1,609	2·63	3,611	2·98	1,903	3·12	5,489	3·50
95	232	1·97	681	2·22	238	2·50	988	3·00
100	15	1·48	64	1·65	—	—	99	2·50
105	—	—	2	1·22	—	—	—	—

* University Research Fellow supported by the Rockefeller Foundation.

whereby each year the number of entrants into the highest aged groups has been successively larger.

Over the period of our inquiry (1931–50) mortality has uniformly decreased, and in some age groups it has decreased very rapidly. Our first question is: has mortality decreased in the older age group? The period 1931–51 is unique in that the only firm estimates of the size of the population are 20 years apart. (The count of the population in 1939 was probably less accurate than a census *sensu stricto*). As is inevitable some error has crept into the annual population figures supplied by the Registrar-General, which are compiled by subtracting deaths and adding new entries into each age group. A check based on the Census population shows this to be true. Since the population estimated in the Census year is certainly more exact than the population estimated in the preceding year, this has been accepted, and graphical estimates of the population have been made for the two years adjoining 1951. The difference in the death rates is appreciable.

Age Group 80 and Over	Year	Registrar-General's Estimated Population	Deaths	Registrar-General's Death Rate	Estimated Correction	Corrected Rate
Males	1950	237,000	44,373	187·2	215,000	206·4
	1951	229,000	49,275	215·2	220,900	223·1
	1952	240,000	45,280	188·7	226,000	200.4
Females	1950	423,000	67,490	159·6	395,000	170·9
	1951	407,000	75,432	185·3	405,900	185·8
	1952	425,000	67,276	158·3	415,000	162·1

These reservations must therefore be borne in mind when an attempt is made to calculate death rates from the published population. Table II gives as moving averages the annual age-specific death rates referable to each sex for the period under discussion. They show that:

(1) During the period 1931–53, there has been a decline in the specific death rates for females in each quinquennium from 55 years of age up to the terminal age group of 80 years and over; but this fall has not been *sustained* in the sense defined in the first communication of this series, since there are small peaks in the period 1944–53;

(2) The same is true of males with two reservations:
(*a*) the proportionate overall decline has been less;
(*b*) there has been no appreciable drop in the quinquennium 60–64.

For the highest age groups our picture is undoubtedly biased by the inaccuracy of annual population estimates. There seems to be evidence (*cf.* Registrar-General's Census Volumes) that population estimates at the end of the period were commonly too high for the old-age groups. So the initial fall and eventual slight rise in the death rates may be to some extent an artefact. This consideration prompts us to compare the Life Tables for the years 1950–52 with similar Tables for 1930–32. This tells us nothing about changes in the intervening period, it does enable us to compare the two terminal triennia referable to census populations; but we cannot yet attain very great accuracy with respect to the latest Census since the only age and sex breakdown so far available is referable to a one per cent. sample. With this reservation we must scrutinize Table I exhibiting an abridged Life Table based on these values.

The difficulty of measuring small survival changes for Scotland in the highest age groups is exemplified in the 1950–52 Life Tables prepared by the Government Actuary, who comments:

> To some extent, therefore, the figures (for Scotland) may be affected by short-term fluctuations from the general secular trend due to the incidence of epidemics or adverse meteorological conditions; at the older ages

TABLE II

TOTAL DEATH RATE

Five-Year Moving Average

Sex	Age	1931–1935	1932–1936	1933–1937	1934–1938	1935–1939	1936–1940	1937–1941	1938–1942	1939–1943	1940–1944	1941–1945	1942–1946	1943–1947	1944–1948	1945–1949	1946–1950	1947–1951	1948–1952	1949–1953
Male	50–54	13·2	13·1	13·3	13·1	13·0	13·4	13·4	13·0	12·8	12·5	11·7	11·3	11·3	10·9	10·8	10·7	10·7	10·6	10·6
	55–59	19·3	19·2	19·4	19·1	19·2	20·1	20·3	19·9	19·9	19·6	18·5	17·9	17·8	17·5	17·4	17·4	17·6	17·5	17·6
	60–64	28·8	28·9	29·3	29·3	29·6	30·7	30·8	30·1	29·9	29·5	28·2	27·6	27·8	27·7	27·8	28·0	28·6	28·4	28·6
	65–69	45·9	45·3	45·1	44·6	44·6	45·8	46·0	45·3	45·1	44·6	42·8	41·8	41·7	41·2	41·4	41·8	43·3	43·5	44·3
	70–74	73·3	73·1	72·9	72·2	72·2	73·4	72·4	70·3	69·2	67·4	64·4	63·0	63·6	63·0	63·8	64·6	66·9	66·8	68·1
	75–79	117·7	117·7	117·6	116·1	117·6	120·8	119·9	116·8	115·5	111·2	104·8	102·0	102·4	100·0	100·7	101·9	105·1	104·8	107·3
	80 and over	204·5	203·4	203·8	200·5	204·4	210·7	209·6	203·1	200·5	192·9	181·4	177·0	180·5	176·2	178·7	181·9	189·2	187·6	193·0
Female	50–54	9·4	9·2	9·2	8·9	8·8	8·9	8·8	8·5	8·3	8·1	7·6	7·3	7·2	6·9	6·8	6·6	6·6	6·4	6·2
	55–59	13·6	13·4	13·2	12·8	12·7	12·9	12·7	12·4	12·2	11·8	11·1	10·7	10·5	10·2	10·1	10·0	9·9	9·7	9·6
	60–64	21·1	20·9	20·9	20·5	20·4	20·7	20·3	19·4	18·9	18·3	17·2	16·7	16·6	16·2	16·1	16·0	15·9	15·6	15·5
	65–69	33·5	33·2	33·0	32·4	32·2	32·8	32·4	31·3	30·9	29·9	28·2	27·2	26·9	26·1	26·0	26·0	26·4	26·0	26·1
	70–74	55·8	55·2	55·1	53·6	53·2	54·2	53·5	51·6	51·0	49·4	46·8	45·7	45·9	44·9	45·3	45·3	45·8	44·8	44·7
	75–79	91·3	90·1	89·7	87·8	88·4	90·4	89·4	86·5	85·5	82·3	77·7	76·0	76·7	74·9	76·0	76·9	78·5	77·2	77·9
	80 and over	177·1	174·6	173·8	167·9	169·9	175·5	173·4	167·3	167·2	161·2	152·4	149·2	151·5	147·3	150·1	153·1	159·7	159·5	164·4

in particular the death rate from respiratory and cardiac diseases can vary considerably from one year to another according to the nature of the weather in the winter months. Nevertheless, it is not to be supposed that if the comparison has been based on the experience of periods other than those used in the construction of Table B the broad picture of secular trend which the table is intended to provide would have differed in any important respect (Government Actuary, 1954).

At the end of the 19th century there had already been a remarkable increase in the expectation of life at birth without as yet any clear indication that the risk of death in the first year of life had begun to lessen. Throughout the first half of the 20th century there has been a sustained and spectacular decline in infant mortality, which so far shows no immediate signs of abatement. Till far into the first half of the 20th century there was no indication of a reduction in mortality at the latter end of life, at or above the 60-year level. With due regard to the sources of error mentioned, it now seems that we have entered a new phase characterized by declining mortality in the terminal age groups. How far this process may continue will be discussed later.

Specific Causes of Death

The incidence of assigned specific causes of death may be scrutinized in two ways. We may exhibit the percentage contribution to total deaths or the actual rate of any one specific disease. Of itself, the elimination of one cause will automatically affect the percentage rating, but not the ranking, of every other cause of death.

Tables III and IV (opposite) show that the alternative methods of actual rate and percentage contribution exhibit most clearly the interplay of different specific causes of death. The Figure illustrates another method; here the actual specific mortality rates are plotted on semi-log paper for each age group. As in previous communications (Taylor, 1954, Taylor and Dauncey, 1954), the use of semi-log paper discloses a straight line as the criterion of a *constant proportionate change*. This procedure shows up most clearly the tempo of death assigned to a particular cause as age advances; but it is only by comparison of the figures with the total death-rate that it is possible to decide whether a specific cause of death is becoming more important *vis à vis* all

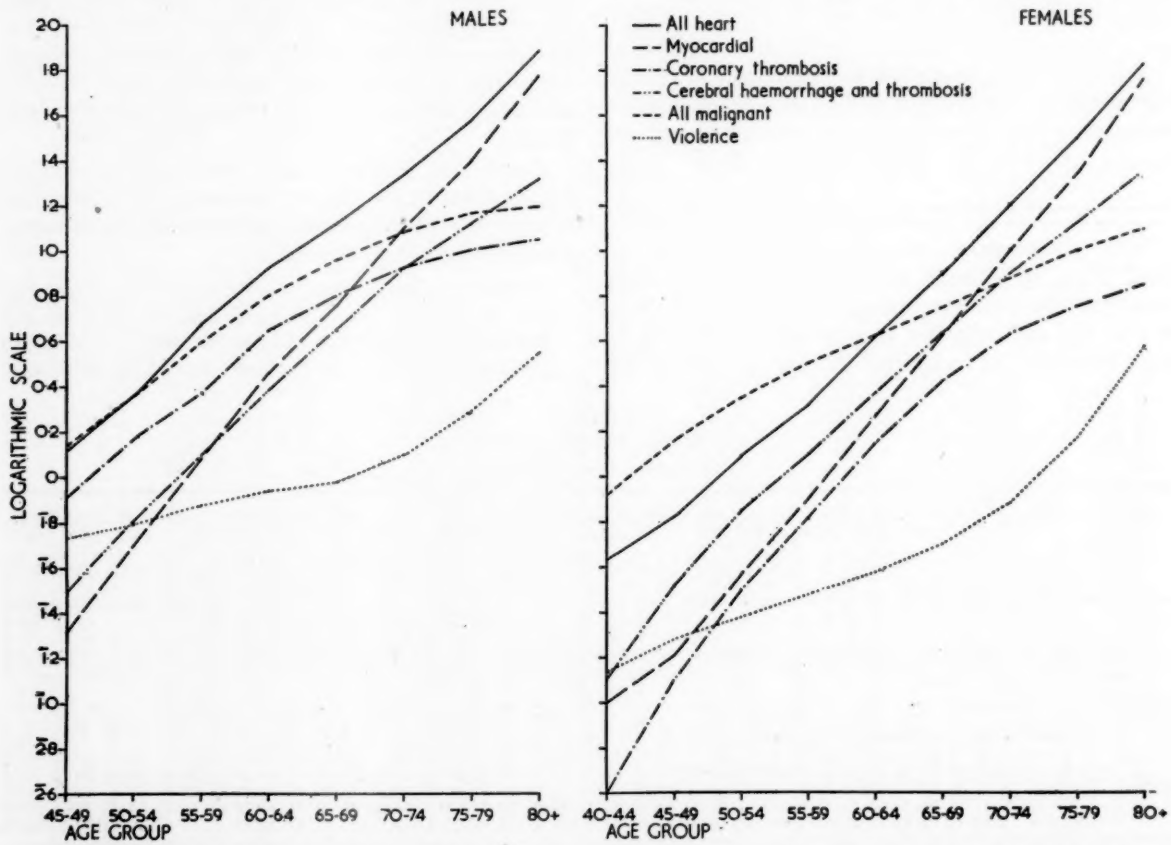

Figure.—Mortality by sex and age group for certain specific causes of death, 1949 (rates per 1,000 population on logarithmic scale).

TABLE III

DEATH RATE PER 1,000 POPULATION (1931 AND 1940)

Sex		Males								Females							
		Age Group								Age Group							
Cause of Death	Year	45–49	50–54	55–59	60–64	65–69	70–74	75–79	80 and Over	40–44	45–49	50–54	55–59	60–64	65–69	70–74	75–79	80 and Over
All Malignant Diseases	1931	1·19	2·10	3·61	5·90	9·09	11·82	13·38	14·03	·99	1·70	2·60	3·54	4·82	6·62	8·80	11·14	12·83
	1949	1·42	2·45	3·99	6·17	8·84	11·99	14·72	15·64	0·84	1·50	2·20	3·14	4·16	5·75	7·76	10·05	12·01
Myocardiac Diseases (1)	*1931*	*0·51*	*0·97*	*1·72*	*3·55*	*6·98*	*13·70*	*24·83*	*47·74*	*0·19*	*0·38*	*0·67*	*1·40*	*2·90*	*5·59*	*11·09*	*20·93*	*43·32*
	1949	*0·22*	*0·51*	*1·22*	*2·98*	*5·93*	*12·60*	*25·32*	*60·48*	*0·10*	*0·17*	*0·38*	*0·80*	*1·85*	*4·20*	*10·30*	*22·20*	*56·91*
Coronary Thrombosis (2)	*1931*	*0·17*	*0·37*	*0·61*	*1·01*	*1·52*	*2·03*	*2·27*	*2·25*	*0·01*	*0·04*	*0·06*	*0·15*	*0·34*	*0·56*	*0·84*	*1·09*	*1·37*
	1949	*0·82*	*1·53*	*2·75*	*4·49*	*6·22*	*8·64*	*10·38*	*11·21*	*0·04*	*0·13*	*0·31*	*0 ·67*	*1·46*	*2·73*	*4·29*	*5·79*	*6·69*
Other Heart Diseases (3)	*1931*	*0·65*	*0·99*	*1·60*	*2·76*	*4·31*	*6·84*	*9·66*	*12·59*	*0·50*	*0·73*	*0·91*	*1·52*	*2·37*	*3·87*	*6·20*	*9·21*	*12·74*
	1949	*0·32*	*0·41*	*0·70*	*1·05*	*1·43*	*2·13*	*2·78*	*3·53*	*0·29*	*0·39*	*0·52*	*0·66*	*0·88*	*1·25*	*1·93*	*2·81*	*3·90*
All Heart Diseases ..	1931	1·33	2·33	3·93	7·32	12·81	22·57	36·76	62·58	0·70	1·15	1·64	3·07	5·61	10·02	18·13	31·23	57·43
	1949	1·36	2·45	4·67	8·52	13·58	23·37	38·48	75·22	0·43	0·69	1·21	2·13	4·19	8·18	16·52	30·80	67·50
Cerebral Haemorrhage and Thrombosis ..	1931	0·24	0·46	0·99	1·79	3·56	6·02	10·02	15·01	0·12	0·29	0·59	0·98	1·73	3·19	5·37	9·05	14·69
	1949	0·31	0·63	1·25	2·59	4·69	8·55	13·54	21·90	0·13	0·34	0·74	1·29	2·33	4·28	8·10	13·76	21·94
All Non-Malignant Respiratory Disorders ..	1931	1·37	1·85	2·30	3·18	4·90	8·14	14·07	29·48	0·44	0·52	0·78	1·17	1·94	3·41	6·51	12·15	26·85
	1949	0·81	1·58	2·87	4·70	6·61	9·15	13·35	23·17	0·26	0·36	0·56	0·94	1·63	2·81	5·22	8·71	18·71
Ulcers of the Stomach or Duodenum	1931	0·37	0·41	0·46	0·47	0·49	0·50	0·51	0·54	0·04	0·09	0·10	0·13	0·13	0·17	0·17	0·23	0·25
	1949	0·25	0·33	0·45	0·61	0·70	0·76	0·90	0·88	0·02	0·04	0·05	0·06	0·13	0·16	0·26	0·31	0·37
Violence	1931	0·88	1·00	1·23	1·32	1·54	1·78	2·57	4·39	0·19	0·24	0·34	0·40	0·51	0·71	1·13	1·95	4·35
	1949	0·55	0·61	0·73	0·89	0·95	1·26	1·95	3·52	0·14	0·19	0·24	0·30	0·38	0·50	0·77	1·49	3·69
Diseases of the Thyroid	1931	0·02	0·02	0·03	0·03	0·04	0·02	0·03	0·03	0·09	0·13	0·15	0·18	0·20	0·22	0·21	0·21	0·16
	1949	0·01	0·02	0·01	0·02	0·02	0·02	0·03	0·01	0·02	0·03	0·04	0·07	0·10	0·14	0·16	0·14	0·10
All Other Causes ..	1931	4·25	5·30	6·91	9·42	15·31	24·42	42·55	86·47	2·48	2·75	3·54	4·73	7·01	10·35	17·13	30·41	69·72
	1949	1·84	2·45	3·51	5·03	7·58	11·58	20·28	44·83	0·99	1·18	1·50	2·06	3·05	4·70	8·11	13·76	33·95
Total Death Rate ..	1931	9·65	13·46	19·47	29·43	47·56	75·27	119·89	212·53	5·05	6·87	9·74	14·20	21·95	34·68	57·45	96·37	186·28
	1949	6·55	10·52	17·48	28·53	42·97	66·68	103·25	195·17	2·83	4·33	6·54	9·99	15·97	26·52	46·90	79·02	158·27

TABLE IV

PERCENTAGE CONTRIBUTION TO TOTAL DEATH RATE OF VARIOUS DISEASES (1949)

Sex	Male								Female							
	Age Group								Age Group							
Cause of Death	45–49	50–54	55–59	60–64	65–69	70–74	75–79	80 and Over	40–44	45–49	50–54	55–59	60–64	65–69	70–74	75–79	80 and Over
All Malignant Diseases	21·7	23·3	22·8	21·6	20·5	18·0	14·3	8·4	29·7	34·6	33·6	31·4	26·0	21·7	16·5	12·7	7·6
Myocardiac Diseases (1) ..	*3·4*	*4·8*	*7·0*	*10·4*	*13·8*	*18·9*	*24·5*	*32·7*	*3·5*	*3·9*	*5·8*	*8·0*	*11·6*	*15·8*	*22·0*	*28·1*	*36·0*
Coronary Thrombosis (2) ..	*12·5*	*14·5*	*15·7*	*15·7*	*14·5*	*13·0*	*10·1*	*6·1*	*1·4*	*3·0*	*4·7*	*6·7*	*9·1*	*10·3*	*9·1*	*7·3*	*4·2*
Other Heart Diseases (3) ..	*4·9*	*4·0*	*4·0*	*3·8*	*3·3*	*3·1*	*2·7*	*1·8*	*10·3*	*9·0*	*8·0*	*6·6*	*5·5*	*4·7*	*4·1*	*3·6*	*2·4*
All Heart Diseases	20·8	23·3	26·7	29·9	31·6	35·0	37·3	40·6	15·2	15·9	18·5	21·3	26·2	30·8	35·2	39·0	42·6
Cerebral Haemorrhage Thrombosis	4·7	6·0	7·2	9·1	10·9	12·8	13·1	11·8	4·6	7·9	11·3	12·9	14·6	16·1	17·3	17·4	13·9
All Non-Malignant Respiratory Disorders	12·4	15·0	16·4	16·5	15·4	13·7	12·9	12·5	9·2	8·3	8·6	9·4	10·2	10·6	11·1	11·0	11·8
Ulcers of the Stomach or Duodenum	3·8	3·1	2·6	2·1	1·6	1·1	0·9	0·5	0·7	0·9	0·8	0·6	0·8	0·6	0·6	0·4	0·2
Violence	8·4	5·8	4·2	3·1	2·2	1·9	1·9	1·9	4·9	4·4	3·7	3·0	2·4	1·9	1·6	1·9	2·3
Diseases of the Thyroid ..	0·2	0·2	0·0	0·0	0·0	0·0	0·0	0·0	0·7	0·7	0·6	0·7	0·6	0·5	0·3	0·2	0·1
All Other Causes	28·0	23·3	20·1	17·6	17·8	17·4	19·6	24·2	35·0	27·3	22·9	20·7	19·2	17·8	17·4	17·4	21·5
Total	100·0	100·0	100·0	100·0	100·0	100·0	100·0	100·0	100·0	100·0	100·0	100·0	100·0	100·0	100·0	100·0	100·0

other causes of death. It is perhaps necessary here to emphasize that the expression *cause of death* has a special connotation in vital statistics since we cannot assign *the* cause and this is particularly true of the aged. Our concern is with a causal nexus, of which one component may have a special interest with regard to the course of action we intend to pursue.

The cause of death recorded in public statistics is what a physician specifies as such on a death certificate to meet the requirements of the law. The causal relevance of this may vary:

(*a*) as standards of differential diagnosis change,

(*b*) as medical educational standards change and with them what practitioners agree to specify as one and the same nosological category.

Moreover, the procedure since 1940 gives the practitioner more scope in assigning the cause of death and the Registrar-General's staff correspondingly less scope to enforce a consistent practice.

As already stated the proper use of such statistics is to enumerate deaths assignable to one among several contributory causes rather than to any unique cause. A specification so conceived has no necessary priority over all others from the viewpoint of aetiology; but it may interest the policy maker in two ways:

(*i*) It may throw light on the efficacy of particular preventive and curative methods and thus indicate a target for further appropriate action.

(*ii*) It may clarify the need for research in particular directions.

A clear appreciation of what we mean by an "assigned cause of death" has a special relevance to our present theme. At the end of the 19th century infant deaths were largely associated with clearly defined communicable diseases amenable to control by preventive methods, and the annual statistics specifying the association of death with any one such disease at least ostensibly gave the policy maker a crude barometer of the success of efforts to control them. At the end of life, the picture is different, how different contemporary interest in gerontology sufficiently reminds us. For example, unless considered as a whole, deaths respectively ascribed to silocosis and to myocardiac failure in 1931 bear no intelligible relation to deaths so designated in 1955.

Pneumoconiosis is of special interest in this context, because it has so recently become a target for legislative action. Before 1925 there was little recognition of the pneumoconioses as a nosological category, and until 1931 they evoked comparatively little attention at an administrative level. Since then various reforms intended to alleviate suffering from industrial disease have drastically altered the value of such death certificates, so that in a rough and ready way, we may distinguish four phases in the vital statistics of the period 1925–55:

(1) practitioners would ascribe nearly all deaths now ascribed to silicosis to myocardiac failure, pneumonia, or tuberculosis;

(2) practitioners might ascribe deaths to silicosis without confirmatory evidence by autopsy;

(3) the Registrar-General's office would exercise its discretion in assigning silicosis as the cause of death if this condition was mentioned on the certificate with myocardiac failure or pneumonia or tuberculosis.

(4) the Coroner's verdict based on the sworn testimony of the pathologist who performed the autopsy, is nearly always the definitive criterion.

From a biological viewpoint, we may, of course, cite silicosis on the one hand or myocardiac failure, pneumonia, or tuberculosis on the other as *a* cause of death; but when preventive action is directed to the elimination of pneumoconiosis or when the community directly or indirectly assumes financial responsibility for its consequences, its specification as *the* cause of death acquires a new significance. If the community decided to take an equally urgent view of asthma as a handicap to national efficiency, the discretion of the physician in citing asthma or myocardiac failure as the cause of death would compromise the sensitivity of the statistical barometer, for the same reason.

Needless to say, myocardiac failure and heart disease in general bulk largely in the vital statistics of the older age groups whence the breakdown of deaths under myocardiac and coronary groups has been the subject of discussion by Ryle and Russell (1949). To make matters more difficult, the diagnostic classification of the General Register Office changed in 1940. Ryle and Russell considered Groups 93 (*b*) and (*c*) together with 97 (3) of the Fourth Revision of the International Classification as definitive of myocardiac diseases for the period 1931–39, but they considered Groups 93 (*c*) and (*d*) of the Fifth Revision as definitive for the period 1940–45. In what follows, the whole of Group 93 has been used. The difference which results is not small at higher ages but it is very small at lower ages. Thus for 1931, the criterion used by Ryle and Russell gives 621 deaths at age 45–49 compared with 603 on the criterion here used; for the terminal age group (80 and over), the two methods give 6,991 and 5,992 male deaths respectively. Ryle and Russell (1949, p. 388) discuss the possibility that there has been a transference from deaths previously ascribed to myocardiac disease to the more specific coronary category, and they come to the following conclusion:

> The increase in the coronary disease mortality in the post-war period is unlikely to be wholly due to transference of myocardiac deaths, because there is evidence that the coronary death-rate itself was increasing less rapidly in this period than in pre-war years—the actual death-rates at ages in 1945 being smaller than those predicted on the basis of the statistical experience relating to the period 1931–39.

It is scarcely necessary to add that similar difficulties are encountered with statistics for hypertension and nephritis. In short, it is not possible to refine further the diagnostic categories for the terminal age groups. The General Register Office has long recognized this dilemma (Registrar-General, 1949).

Breakdown by Diagnosis in the Terminal Age Groups

A breakdown by diagnosis of the major diseases contributing to mortality from 45 years of age onwards discloses a comparatively small group of categories contributing to over 50 per cent., and in the age of groups above 50 years to well over 60 per cent. of all deaths. Tables III and IV and the Figure summarize the relevant data; they show that the changing picture reveals no clear-cut demarcation of an old-age group. On the other hand, we can broadly distinguish between three categories, which for males are clearly defined:

(*a*) for malignant diseases and coronary thrombosis the annual incidence falls off sharply at about 75 years of age:

(*b*) for myocardiac diseases, cerebral thrombosis or haemorrhage, and respiratory disorders (not illustrated) the death rate remains fairly constant from 45 years to the end of life; for myocardiac disease the incidence increases until the final age group.

(*c*) for violence (including accidents, etc.) the death rate rises sharply at about 65 years of age.

This three-fold distinction is also roughly applicable to females; but the fall-off for malignant diseases and for coronary thrombosis about 75 years of age is much less striking.

Sex Differences in Ageing.—The relative incidence of death from malignant disease and heart disease shows a clear sex differential. For 1949 (Table IV) the percentage contribution to all deaths for these two major causes shows the same clear shift of emphasis for each sex at a different critical age.

For males aged 45–49 the malignant diseases are slightly more important as a major cause of death; at age 50–54 the figures for the two groups of disease are equal; thereafter the heart diseases are in the ascendant, and in the final age group (80 years and over) they are almost five times as important.

For females the changeover comes 10 years later. Not until age 60–64 do deaths from malignant disease equal the ever-increasing deaths from heart disease. However, the heart diseases in the final age group are five and a half times as important as the malignant diseases.

If there is to be any great improvement it must clearly come about by a decrease in the deaths now ascribed to the heart diseases.

A similar 10-year sex differential is also apparent in deaths from other causes. In Table II a small retrogression in the total death rate is first apparent for males aged 55–59 in the quinquennium 1947–51. A similar retrogression in the female death rate in the same quinquennium appears 10 years later in life, at age 65–69. Our data throw no light on how far this 10-year difference in ageing is due to social circumstances and how far to innate agencies.

The Prospect for Conservation of the Aged

For reasons stated above, the breakdown summarized in Tables III and IV is grossly and advisedly coarse, because so many of the major contributory "causes" of deaths certified as such refer to diagnostic categories of dubious value. On this understanding, and if we also grant that present indications of a slight sustained decline of mortality in the terminal age groups are not illusory, we may essay a tentative opinion on the limits that may be set to such a decline in the absence of further relevant advances of medical knowledge. Before doing so, three considerations call for comment:

(*a*) We have not as yet assessed how far a healthier start in life protects the individual surviving to a late age against the hazards incident thereto;

(*b*) Since the major sources of wastage were formerly epidemic diseases which presented a challenge less to medical research than to administrative action, a contemporary forecast for the aged is not comparable to the prospect for reduction of infant mortality when the first indications of a decline were detectable 50 years ago;

(*c*) Since the label attached to a very large number of deaths ascribed to "myocardiac failure" merely records the final scene of a drama the first act of which we have not witnessed, we cannot say what the course of events would have been if earlier treatment of now remediable or preventable disorders had been available at the appropriate time.

Preventive measures *may* accomplish an appreciable reduction of deaths classified as due to "violence", and domiciliary curative measures for pneumonia *may* continue to reduce the deaths directly attributable to respiratory disorders. Earlier diagnosis and treatment of peptic ulcer and the use of anticoagulants for coronary or cerebral thrombosis *may* conceivably accomplish a little; but our present slight knowledge of hypertension, cerebral haemorrhage and the overwhelming bulk of insidious malignant disease offers little hope of immediate improvement. We cannot therefore predict a considerable decline in mortality in the late age groups unless substantial advances in medical science occur *pari-passu*. Neither should we dismiss the possibility out of hand. A confident

forecast would be justifiable only if we could assess how far protection from risks to health in early life may reduce the risk of death in the terminal years. Here the cohort method of analysis may throw some light on an otherwise obscure situation; the concluding paper in this series will disclose what further clues this method can reveal.

Summary

(1) Annual age-specific death rates for the period 1931–53 show a decline for both sexes and for all age groups, with the single exception of males aged 60–64. The population estimates over this period are, however, suspect, and comparison by Life Tables based on the Census population are necessarily approximate since the final 1951 Census Population is not yet available. With these considerations in mind, there appears to be evidence of declining mortality in the terminal age groups.

(2) In the old there is rarely a single *cause* of death, and during this period the relevance to vital statistics of the cause stated on the death certificate has varied. A proper use of statistics based on information supplied by death certificates is to enumerate deaths assigned to causes relevant to social policy. In the later age groups, practitioners are dealing with disorders about which little is known or what is known has become known very recently. The resulting changes in classification over a period of years is illustrated by the history of the pneumoconioses, and the changes in classification of heart diseases. Therefore it is not possible to refine further the diagnostic categories for the terminal age groups.

(3) The breakdown by diagnosis of deaths of persons over 50 years of age shows that malignant diseases, heart diseases, and non-malignant respiratory diseases contribute to over 60 per cent. of all deaths. There is a sex difference in the relative importance at different ages of the two major causes, malignant diseases and heart diseases.

(4) No confident prediction of prospects for the conservation of the aged can be made from analysis of the annual rates.

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